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A RING-SPOT TYPE OF VIRUS DISEASE OF TOMATO[1]

R. W. Samson[2] and E. P. Imle[3]

(Accepted for publication May 5, 1942)

A virus disease of the tomato, characterized, in the most conspicuous stages, by intricate patterns of necrotic rings and lines on young leaves and necrotic streaks on the stems of infected plants, occurs in field and garden plantings throughout Indiana. Fruits faintly to conspicuously marked with concentric, brown, corky, necrotic rings of varying size and extent sometimes occur on the diseased plants. The disease was first noted in July, 1930, at Vincennes, Indiana, and has since been observed on as high as 90 per cent of the plants in many canning-tomato fields in the southern part of the State. Scattered infected plants have been noted as early as mid-July, but the disease is most abundant on field tomatoes in August and September. Its occurrence in Missouri has been confirmed by inoculations from several collections of diseased tomatoes received through the courtesy of C. M. Tucker of the Missouri Agricultural Experiment Station. A single collection was made in Illinois in August, 1941. The virus has been secured from vaguely mottled plants of horse nettle (*Solanum carolinense* L.) growing in tomato fields, indicating that this may be a commonly occurring perennial weed host. Plants of *Nicandra physalodes* (L.) Pers. and *Datura stramonium* L. infected with the virus have been found in and near tomato fields. The disease must be somewhat destructive to the tomato crop, but the extent of damage has been difficult to estimate because the extensive epidemics so far observed have been coincident with serious defoliation caused by *Septoria lycoperisici* Spegg. and *Alternaria solani* (E. and M.) Jones and Grout. The effects of the fungous diseases have overshadowed those of the virus.

The necrotic rings produced on the fruit, foliage, and stems of tomato and on the leaves of other hosts suggest the name, tomato ring spot, for the disease. Only brief references to the writers' work on it have been published previously (3, 11).

SYMPTOMS OF RING SPOT ON TOMATO

The conspicuous feature of a tomato plant naturally affected with ring spot in the field is the curling and extensive necrosis of the terminals of one or more actively growing shoots. Brown, clearly defined, necrotic rings and sinuous lines appear on the basal portions of leaflets of the younger leaves (Fig. 1, A). The petioles of the necrotic leaves and the adjacent portions of the stems are frequently marked by necrotic streaks and rings. Under greenhouse conditions, small, brown, necrotic rings may appear within 6 to

[1] Journal Paper No. 24, of the Purdue University Agricultural Experiment Station, Lafayette, Indiana. Contribution from the Department of Botany.

[2] Assistant Chief in Botany, Purdue University Agricultural Experiment Station.

[3] Formerly Graduate Assistant in Botany, Purdue University Agricultural Experiment Station.

12 days on inoculated leaves of young tomato plants, but such local lesions do not always result. Three to 6 days later the typical ring-and-line patterns appear on the young leaves and stem tips, sometimes in sufficient intensity to kill the terminals. The length of the incubation period is less at 70° and 80° than at 62° F. Resumption of growth of the terminals or of one or more lateral buds of affected shoots usually takes place promptly after the development of the necrotic phase of the disease, both under greenhouse and

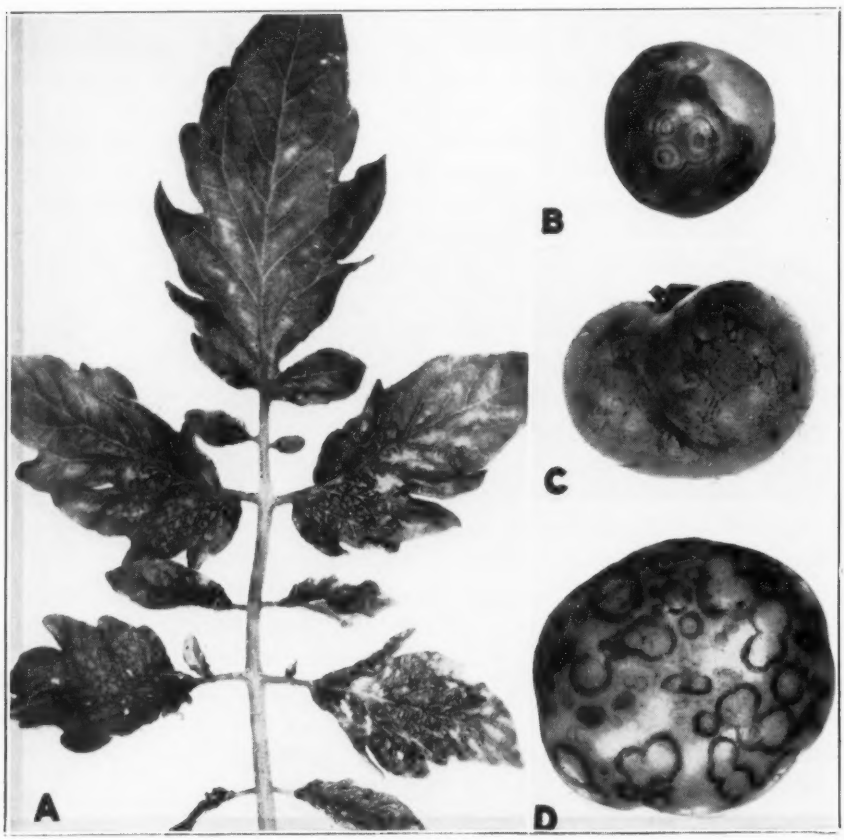

FIG. 1. Tomato ring spot on leaf and fruits of tomato. A. Network of brown, necrotic lines on basal portions of leaflets. B–D. Faint to conspicuous grey or brown, corky, superficial, concentric rings or portions of rings on green fruits.

outdoor conditions. Such new growth shows either no symptoms or only a vague mottling, and appears to develop and fruit normally.

The symptoms that sometimes occur on fruits in the field vary from faint to conspicuous grey or brown, corky, superficial, frequently concentric rings or portions of rings (Fig. 1, B–D). They apparently develop only on those fruits that are very young at the time of virus invasion. They are suggestive of the fruit symptoms described by Samuel, Bald, and Pittman (12) and Parris (8) for the Australian spotted wilt disease, and Milbrath (7) for the

tip blight disease, but differ in being superficial and corky in nature. The presence of the ring-spot virus has been demonstrated repeatedly in ring-marked fruits and symptomless fruits from diseased plants. Symptoms have not yet been secured on fruits of plants artificially inoculated with ring-spot virus, either in the greenhouse or field, despite numerous attempts.

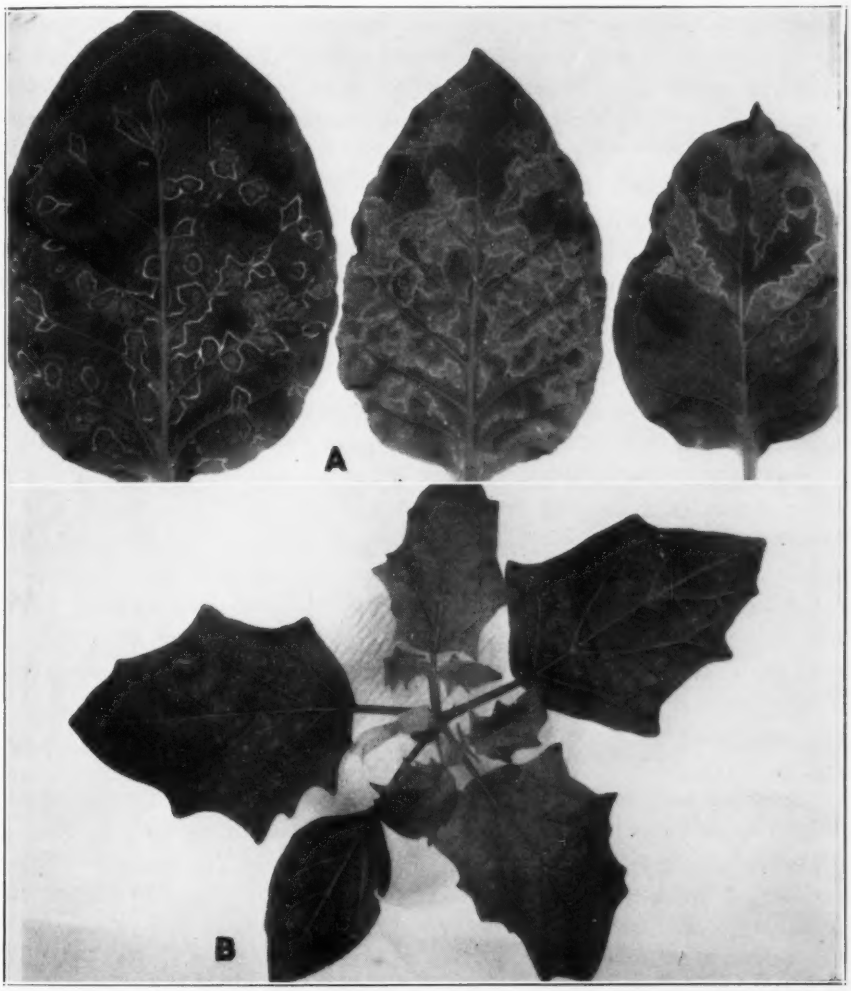

FIG. 2. Tomato ring spot on Turkish tobacco (*Nicotiana tabacum* L.) and jimson weed (*Datura stramonium* L.). A. Conspicuous ring-and-line patterns on tobacco leaves. B. Clearing and slight necrosis of veins, flecking, and inconspicuous ring-and-line patterns on leaves of jimson weed.

SYMPTOMS OF TOMATO RING SPOT ON TOBACCO AND JIMSON WEED

Young plants of Turkish tobacco (*Nicotiana tabacum* L.), infected with tomato ring-spot virus, exhibit a mild ring-and-line pattern on the inoculated leaves and somewhat more conspicuous patterns on one or more leaves next

above (Fig. 2, A). Subsequent growth on such plants may show no such patterns but only a slightly paler color and a somewhat more rigid appearance than normal. Presence of the virus in both the basal and terminal leaves of such plants has been demonstrated by return inoculations to tomato and jimson weed.

Symptoms of tomato ring spot on *Nicotiana affinis* T. Moore consist of numerous, faint, small rings on the lower leaves only, although the virus may be recovered by inoculations from the terminal leaves of such plants. No symptoms of local or systemic infection have appeared on plants of *N. glutinosa* L. or *N. sylvestris* Speg. and Comes inoculated with tomato ring spot.

Indefinite rings and lines appear on the inoculated leaves of young jimson weed plants (*Datura stramonium*), followed by a clearing of the veins, flecking, and rather inconspicuous ring-and-line patterns on the next younger leaf tissues (Fig. 2, B). Subsequent leaf growth is almost devoid of any symptoms of disease, although definitely invaded by the virus.

TRANSMISSION OF TOMATO RING SPOT

Some difficulty was encountered in transmitting tomato ring spot to various hosts by leaf rubbing, unless carborundum powder was applied to the leaves prior to inoculation. The virus is easily thus transmitted to jimson weed. Several hundred attempts, all unsuccessful, were made to transmit the earlier collections of ring spot in plant extract from this host back to tomato. They were, however, transmitted with ease to tobacco, thence back to tomato. They also could be transmitted directly from jimson weed to tomato by grafting. Later, several collections were obtained that could be transmitted directly from jimson weed to tomato in plant extract without intermediate transfer through tobacco.

FAILURE TO TRANSMIT RING SPOT WITH THRIPS

Attempts were made to transmit tomato ring spot with thrips (apparently, *Thrips tabaci* Lind.). Thrips were confined for periods of 10 days to 9 weeks on virus-infected tomato and jimson weed. The insects were then transferred to feeding traps on the leaves of healthy plants of tomato and jimson weed and fed for varying intervals. In some instances they were transferred successively to 4 to 6 new plants after several hours of feeding on each plant in the series. None developed ring spot. The 9-week period should have been long enough for a new generation of thrips to have developed and become viruliferous (6, 10).

Parallel attempts to transmit a suspected culture of the spotted-wilt virus with the same stock of thrips used in the ring-spot inoculations were likewise unsuccessful.

HIGH TEMPERATURES FAVOR DEVELOPMENT OF TOMATO RING SPOT

Relatively high greenhouse temperatures favor the development of ring

spot on tomato. Two different experiments were performed wherein lots of 20 plants were inoculated and placed in each of 3 greenhouse sections maintained at approximately 62°, 70°, and 80° F., respectively. The disease appeared at about the same time on the plants at the two higher temperatures and much more quickly than at 62° F. It was most severe on the plants in the 80° section, killing 75 per cent of the plants in one of the experiments.

Infected tomato plants, grown in a glass-enclosed compartment held at a constant temperature of 88° F., reacted differently, however; becoming stunted and marked with an unusual yellow mosaic pattern. Under these conditions infected jimson weed developed only a few pale rings on the inoculated leaves but no further symptoms. After one month in this compartment the plants were removed to a greenhouse section held at 68° F. Necrotic ring spots developed on young leaves and shoots of the tomato plants after 30 days, while the jimson weeds showed no further evidence of disease. The virus was subsequently recovered from the younger tissues of both species.

HOST RANGE OF TOMATO RING SPOT

Tomato ring spot has been found in the field on the varieties Greater Baltimore, Century, Clark's Early, Kalb, King Humbert, Marglobe, Pride of Illinois, Pritchard, and Rutgers. The varieties Comet, Early Detroit, Earliana, Gulf State Market, Oxheart, and South Australian Dwarf Red were successfully inoculated with the virus.

Seventy-eight species, representing 27 plant families, were tested for susceptibility to tomato ring spot. Five plants were inoculated from 2 or 3 tomato plant sources and from one jimson weed source, making a total of 15 or 20 inoculated plants of each species or variety. Return inoculations were made to tomato and jimson weed to determine systemic infection, regardless of whether the test plants showed symptoms of disease or not. Symptoms were secured on the following 2 species of the Amaranthaceae, one of the Martyniaceae, and 19 species or varieties of the Solanaceae:

Family	*Genus and Species*	*Common Name*
Amaranthaceae	*Amaranthus tricolor* L.	
	A. retroflexus L.	Pigweed
Martyniaceae	*Martynia louisiana* Mill.	Martynia
Solanaceae	*Androcera rostrata* (Dunal) Rydb.	Buffalo burr
	Capsicum frutescens L.	Pepper, Ruby King
	Datura stramonium L.	White flowered jimson weed
	D. tatula L.	Purple flowered jimson weed
	Datura sp. (horticultural form)	Vaughan's variety, Cornucopia
	Hyoscyamus niger L.	Henbane
	Lycopersicon esculentum Mill.	Tomato, 10 varieties
	L. pimpinellifolium (Jusl.) Mill.	Red currant tomato
	Nicandra physalodes (L.) Pers.	Apple of Peru
	Nicotiana tabacum L., var. Turkish	Tobacco

Genus and Species	*Common Name*
N. affinis T. Moore	
Petunia hybrida grandiflora Vilm.	Giant Ruffled Petunia
P. hybrida Vilm.	Howard's Star Improved Petunia
P. axillaris BSP.	Petunia
Physalis alkekengi L.	Chinese lantern plant
Solanum melongena L.	Eggplant
S. pseudo-capsicum L.	Jerusalem cherry
S. tuberosum L.	Potato
S. carolinense L.	Horse nettle

The ring-spot virus was recovered from all of the above by return inoculation to jimson weed and tomato, except from *Amaranthus retroflexus, Martynia louisiana, Capsicum frutescens, Hyoscyamus niger,* and *Solanum pseudo-capsicum.*

The following species failed to develop symptoms or to yield the ring-spot virus on return inoculation to jimson weed and tomato:

Family	*Genus and Species*	*Common Name*
Aizoaceae	*Tetragonia expansa* Murr.	New Zealand spinach
Apocynaceae	*Vinca rosea* L., 2 varieties	Periwinkle
Asclepiadaceae	*Asclepias tuberosa* L.	Milkweed
Begoniaceae	*Begonia semperflorens* Link & Otto	Begonia
Chenopodiaceae	*Chenopodium album* L.	Lamb's quarter
	Beta vulgaris L., 2 varieties	Garden beet
	B. vulgaris var. *cicla* L.	Swiss chard
Compositae	*Ambrosia artemisiifolia* G.	Ragweed
	Zinnia elegans Jacq., 2 varieties	Zinnia
	Bidens bipinnata L.	Spanish needle
	B. discoidea (T. & G.) Britton	Spanish needle
	Emilia sonchifolia DC.	Tassel flower
	Helianthus annuus L.	Sunflower
	Erigeron speciosus DC.	Erigeron
	Calendula officinalis L., 2 varieties	Calendula
	Callistephus chinensis Nees.	China aster
	Tagetes signata Bartl.	Marigold
	T. erecta L.	Marigold
Convolvulaceae	*Ipomoea batatas* Lam.	Sweet potato
	I. purpurea Lam.	Morning glory
	Quamoclit penata Bojer, 3 varieties	Cypress vine
Cruciferae	*Brassica oleracea* L., 2 varieties	Cabbage
	B. alba Rabenh., 2 varieties	Mustard
	B. rapa L., 2 varieties	Turnip
Cucurbitaceae	*Lagenaria leucantha* Rusby	Dipper gourd
	Luffa cylindrica Roem.	Dish cloth gourd
	Cucurbita pepo L., 3 varieties	Nest egg gourd, Golden Summer Crookneck squash, Cocozelle Bush squash
	C. moschata Duchesne	Cushaw pumpkin
	C. maxima Duchesne	Hubbard squash
	Cucumis melo L., 2 varieties	Cantaloupe
	C. sativus L., 2 varieties	Cucumber
	C. anguria L.	West Indian gherkin
	Citrullis vulgaris Schrad., 2 varieties	Watermelon
Dipsaceae	*Scabiosa atropurpurea* L.	Scabiosa
Euphorbiaceae	*Euphorbia heterophylla* L.	Mexican fire plant

Family	*Genus and Species*	*Common Name*
Geraniaceae	*Pelargonium hortorum* Bailey	Pelargonium
	P. odoratissimum Ait.	Pelargonium
Leguminosae	*Lupinus hartwegii* Lindl.	Lupine
	Phaseolus vulgaris L.	Golden Cluster wax bean
	P. limensis Macf.	Lima bean
	Glycine max Merr., 2 varieties	Soybean
Malvaceae	*Abutilon theophrasti* Medic.	Indian mallow
	Hibiscus esculentus L.	Okra
Phytolaccaceae	*Phytolacca decandra* L.	Pokeweed
Plantaginaceae	*Plantago lanceolata* L.	Bracted plantain
	P. major L.	Common plantain
Polemoniaceae	*Phlox drummondii* Hook., 2 varieties	Phlox
Polygonaceae	*Rumex acetosella* L.	Red sorrel
	Pesicaria hydropiperoides Michx.	Smartweed
Ranunculaceae	*Delphinium ajacis* L., 2 varieties	Larkspur
Scrophulariaceae	*Antirrhinum majus* L., 2 varieties	Snapdragon
	Verbascum thaspus L.	Mullein
Solanaceae	*Lycopersicon peruvianum* (L.) Mill.	
	Nicotiana glutinosa L.	
	N. sylvestris Speg. & Comes	
	Salpiglossus sinuata L., 2 varieties	Salpiglossus
Tropaeolaceae	*Tropaeolum majus* L., 2 varieties	Nasturtium
Umbelliferae	*Daucus carota* L.	Carrot
Violaceae	*Viola tricolor* L., 2 varieties	Pansy

PROPERTIES OF THE TOMATO RING-SPOT VIRUS

Tolerance to Heat. Two milliliter portions of infectious tomato juice, filtered through cheesecloth, were placed in small, thin-walled test tubes and suspended for 10-minute periods in a water bath at various temperatures; they were then immediately inoculated into young tomato plants. Ring-spot infection was secured from preparations heated at 56° but not at 58° C. or higher.

Resistance to Aging in Vitro. Quantities of juice from ring-spot-infected tomato plants held in test tubes at laboratory temperatures were infectious after 21 hours, but not after 27 hours, when inoculated into jimson weed. Tomato plants were infected from such juice stored for 21 hours at 3° C. but only jimson weed plants were infected with juice stored for 45 hours at this temperature. Complete inactivation occurred after 60 hours storage at 3° C.

Limits of Dilution. The tomato ring-spot virus in tomato juice was found to be infectious only at relatively low dilutions. Preparations diluted to 1:250 were infectious while those diluted to 1:500 were not.

Desiccation of the Ring-spot Virus in Infected Plant Tissue. Ring-spot infection was secured from water extracts of tomato leaves dried in the light in a warm greenhouse for 114 hours but not for 300 or 530 hours. The virus was destroyed more quickly by drying in tobacco leaves than in tomato leaves.

COMPARISON OF TOMATO RING SPOT WITH SPOTTED WILT, TOBACCO RING SPOT, POTATO RING SPOT, AND RING MOSAIC

Spotted Wilt. Differences in symptoms produced, properties, and host range distinguish the tomato ring-spot virus from the spotted-wilt virus. The writers have compared three different collections of spotted wilt with tomato ring spot in the greenhouse. The symptoms produced on tomato by these collections have been in almost complete agreement with those described by Samuel, Bald, and Pittman (12).

Bronzing is not a characteristic symptom of tomato ring spot on tomato. The necrotic symptoms of ring spot on tomato fruits (Fig. 1, B–D) seem to be more clearly defined, when they do occur, than those described by Samuel, *et al.*, (12) and Parris (8) for spotted wilt.

The tomato ring-spot virus survives a temperature of 56° C. for 10 minutes, as compared to inactivation of the spotted wilt virus at 42° C. (1). The tomato ring-spot virus survives aging *in vitro* for 21 hours at room temperatures, while the spotted wilt virus is reported to lose its infectiousness after 6 hours at such temperatures (1). This, however, may not be a sufficient difference in tolerance to aging to distinguish the two viruses.

The host range of tomato ring spot does not appear to be as extensive as that of spotted wilt. *Calendula officinalis, Zinnia elegans, Scabiosa* sp., *Lupinus* sp., *Phaseolus vulgaris, Plantago major, Delphinium* sp., *Antirrhinum majus, Nicotiana glutinosa, N. sylvestris, Salpiglossus* sp. and *Tropaeolum majus* are listed as hosts of the spotted-wilt virus (13). None of these were successfully infected with the ring-spot virus.

Parris (8) and Sakimura (10) present evidence that a virus causing a ring spot of tomato is the same as that of pineapple yellow spot and spotted wilt. The host range, symptoms produced, thermal death point and transmission by *Thrips tabaci* indicate the tip-blight virus, described by Milbrath (7), to be very similar to, if not identical with, the spotted-wilt virus and distinct from tomato ring spot.

One cross-protection test was made, in which 8 rooted and rapidly growing cuttings from tomato plants infected with the tomato ring-spot virus were inoculated with a suspected culture of the spotted-wilt virus. Typical symptoms of spotted wilt appeared on all cuttings 7 days after inoculation. That all of the cuttings were infected with tomato ring spot, even though none showed any symptoms, was demonstrated by inoculating young seedlings from them prior to inoculation with spotted wilt. The spotted-wilt virus was secured from a diseased tomato plant received from L. J. Alexander of the Ohio Agricultural Experiment Station, who collected it in an Ohio greenhouse. Attempts to transmit it with the onion thrips failed, but otherwise it agreed with published descriptions of spotted wilt (8, 12, 13). It was transmitted to tomato, jimson weed, Turkish tobacco, *Nicotiana glutinosa, Emilia sonchifolia,* aster, zinnia, snapdragon and nasturtium, with the development of typical symptoms. In parallel inoculations only the first three of the above were successfully infected with the tomato ring-spot virus used to inoculate the tomato plants from which the cuttings were taken.

Tobacco Ring Spot. Differences in transmissibility, symptoms produced and host range serve to distinguish the tomato ring-spot virus from the tobacco ring-spot virus (9, 14). The ring-and-line patterns usually produced by it on Turkish tobacco are broader and more severe in their effects than those produced by the tomato ring-spot virus. Tobacco ring spot has been transmitted to such species as *Tetragonia expansa, Beta vulgaris, Chenopodium album, Ambrosia artemisifolia, Bidens discoidea, Calendula officinalis, Helianthus annuus, Tagetes erecta, Zinnia elegans, Ipomoea purpurea, Citrullis vulgaris,* various species of *Cucumis, Scabiosa atropurpurea, Phaseolus vulgaris, Phytolacca decandra, Nicotiana glutinosa,* and *Antirrhinum majus,* (14), none of which could be infected with the tomato ring-spot virus.

Price (9) compared the host ranges of 6 plant viruses, one of which he refers to as tomato ring spot. He reports *Tetragonia expansa, Vinca rosea, Beta vulgaris, Helianthus annuus, Zinnia elegans, Cucumis sativus, Pelargonium hortorum, Phaseolus vulgaris, Phytolacca decandra, Phlox drummondii, Nicotiana glutinosa* and *N. sylvestris* as susceptible to this virus. No symptoms were produced on any of these hosts by the writer's tomato ring-spot virus, nor could it be recovered from any of them. Symptoms produced by a culture of Price's tomato ring spot on tomato, jimson weed and Turkish tobacco were strikingly different from those produced by the writer's tomato ring spot.

Potato Ring Spot. The tomato ring-spot virus commonly occurs in tomato plants in the field showing symptoms of tobacco-mosaic infection. This suggested that the disease might be due to the combined effects of the two viruses, or that the tomato ring-spot virus might be the same as the potato ring-spot virus (5), which acts in complementary fashion with the tobacco-mosaic virus to produce the necrotic disease, tomato streak (2, 15). Since the tobacco-mosaic virus does not become systemic in *Datura stramonium,* it has been possible to inoculate this plant from tomato plants showing symptoms of both ring spot and tobacco mosaic, and subsequently transfer the ring-spot virus, freed of tobacco mosaic, back to tomato. Typical ring-spot symptoms have been secured repeatedly on tomato plants so inoculated. Likewise, the disease has been obtained, free of tobacco mosaic, on several occasions by direct inoculation from naturally infected tomato and *Solanum carolinense* plants.

Tomato plants infected with, and showing symptoms of potato ring spot are not protected against subsequent infection by tomato ring spot. Typical symptoms of the latter have been secured on such tomato plants.

Ring Mosaic. Johnson (4) described a ring mosaic of tobacco that produces concentric, sunken, chlorotic rings on tomato fruits and streaking of the stems. The fruit symptoms are not characteristic of tomato ring spot. The other symptoms produced by the ring-mosaic virus on tomato, the resistance of the virus to drying, and the fact that it does not become systemic in jimson weed further distinguish it from the tomato ring-spot virus.

SUMMARY

A virus disease of the tomato, characterized by intricate patterns of necrotic rings and sinuous lines on young leaves, streaks on the stems, and faint to conspicuous, concentric, brown, necrotic rings of varying size and extent on the fruit, occurs in field and garden plantings throughout Indiana. The symptoms produced on tomato and other hosts suggest the name, tomato ring spot, for the disease.

Infected tomato plants outgrow the necrotic phase of the disease and subsequently show either no detectable symptoms or only a vague mottling. The virus is present in the symptomless parts of such plants.

Attempts to transmit tomato ring spot with *Thrips tabaci* Lind. (onion thrips) were unsuccessful and inconclusive.

Under greenhouse conditions the disease develops more rapidly and is more destructive at a temperature of 80° than at 70° or 62° F.

Seventy-eight species, representing 27 plant families, were tested for susceptibility to tomato ring spot. Disease symptoms were produced on two species of the *Amaranthaceae*, one of the *Martyniaceae*, and on 19 species and varieties of the *Solanaceae*. It was recovered, by return inoculation to jimson weed and tomato, from *Amaranthus tricolor* and from 16 species and varieties of the *Solanaceae*.

Studies of the properties of the virus in tomato-plant extract indicate that: (a) its thermal death point is between 56° and 58° C., (b) it survives aging *in vitro* for 21 hours but not 27 hours at room temperatures, and for 45 but not 60 hours at 3° C., (c) it is infectious at dilutions of 1:250 but not 1:500, and (d) it survives desiccation in detached tomato leaves exposed in a warm greenhouse for 114 but not 300 hours.

Differences in symptoms of disease, host ranges, and properties clearly indicate that the tomato ring-spot virus is distinct from the tobacco ring-spot, potato ring-spot, and ring-mosaic viruses. Symptoms produced by it on tomato are highly suggestive of those produced by the spotted-wilt virus on this host, but it has a more restricted host range and a higher thermal death point. Tomato plants affected with tomato ring spot were not protected from infection by a suspected culture of the spotted-wilt virus.

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RELATIVE RESISTANCE OF ALFALFA SPECIES AND VARIETIES TO RUST CAUSED BY UROMYCES STRIATUS[1]

J. M. KOEPPER[2]

(Accepted for publication March 30, 1942)

INTRODUCTION

Alfalfa rust (*Uromyces striatus* Schroet.) is widely distributed in the United States, but usually has been considered one of the minor diseases. In recent years, however, it has wrought considerable damage, especially to the seed crop, in parts of central United States. The only practical means of control seems to be the breeding of resistant varieties through selection and hybridization. The studies herein reported were undertaken to obtain information on methods of culturing alfalfa rust in the laboratory, greenhouse, and nursery and to test varieties, selections, and species of alfalfa in search of sources of resistance.

This disease is of little economic importance in some regions, but in other areas it is regarded as one of the most important leaf and stem diseases. During the last 25 years, 1916 to 1940, inclusive, alfalfa rust was reported 29 times, either in the supplements or in the regular numbers of the *Plant Disease Reporter* (1, 5, 6, 7). Workers report varying degrees of damage, depending mainly on the region in question and the prevailing environmental conditions. Damage in any one season depends largely upon the amount of rainfall during the summer months and the later part of the growing season. In nearly all cases alfalfa rust seems to do its greatest damage to the crop that is being grown for seed. Under favorable conditions, however, it may be conspicuous in the later hay cuttings in the western Mississippi valley. From all indications the disease produces greater losses in the warm, humid parts of the United States than in the cooler areas.

Few reports were found in the literature on resistance to alfalfa rust. During the summer of 1911, plants in the Iowa Experiment Station plots were examined (4). One plot of an unnamed selection with nearly white flowers was found severely rusted, but an adjoining plot of a recent introduction from Germany was sparsely infected. In 1922 several plants in a severely infected field of alfalfa growing on the Soils and Crop Experiment Farm at Lafayette, Indiana, showed high resistance (3). These were transplanted to the greenhouse, where their rust resistance was proved by inoculation. Mains (2) stated in 1926 that plants resistant to alfalfa rust had been discovered in strains of such varieties as Grimm, Cossack, Vale, Argentine, Dakota, and New Zealand.

[1] Contribution No. 416 from the Department of Botany, Kansas Agricultural Experiment Station, Manhattan, Kansas.

[2] Formerly, Graduate Research Assistant, Kansas Agricultural Experiment Station, Manhattan, Kansas. The writer wishes to acknowledge his indebtedness to C. O. Johnston and L. E. Melchers for valuable assistance and direction throughout the investigation.

EXPERIMENTAL RESULTS

In a study of varietal resistance in the laboratory, excised alfalfa leaves lived 3 to 4 weeks in moist Petri dishes. Inoculating leaflets by applying fresh urediospores with a small camel-hair brush gave excellent infection (Fig. 1). Rust pustules appeared on leaves inoculated in this manner 2 to 3 days earlier than on potted plants placed in a moist chamber in the greenhouse and inoculated with a spore suspension. This method of inoculation proved sufficiently accurate to determine varietal and species differences in susceptibility.

FIG. 1. Normal uredia of *Uromyces striatus* produced by the inoculation of excised leaves of Turkestan alfalfa in the laboratory.

Several methods of inoculation and incubation were tried in an effort to produce infections in the greenhouse comparable to those obtained in the field. Most methods either were too tedious or gave poor results, but after numerous trials a simple method was discovered, which, if conducted in the proper manner, yielded better results than field tests. It was found that fresh urediospores must be used to obtain satisfactory infection. A composite inoculum was prepared by scraping urediospores from infected leaves with a flattened needle. This inoculum was suspended in distilled water in the glass container of a small fly sprayer and atomized on potted alfalfa plants in moist chambers. The relative humidity was retained at close to 100 per cent for 48 hours; then the plants were removed and placed on wet sand beds on the floor of the greenhouse. Rust pustules appeared 7 to 10 days after inoculation, and final rust readings generally were taken about 3 weeks later.

TABLE 1.—*Description of infection types used in recording reaction of alfalfa varieties and species to Uromyces striatus in the greenhouse*

Host reaction	Numerical equivalent	Coefficient of severity	Type of infection	Length of infection period
Immune	0	No macroscopic evidence of infection		
Highly resistant	1	Uredia none or very few, 1 to 3 per leaflet; small, fleck-like.	Uredia, when present, non-erumpent and always in necrotic areas. Occasionally necrotic areas produced without development of uredia. Pathogen dies.	No macroscopic evidence of infection until 2 weeks after inoculation; no apparent damage to plant at 4-week period.
Resistant	2	Uredia small, scattered, 3 to 10 per leaflet.	Uredia slightly erumpent, generally surrounded by or within a necrotic or very chlorotic area.	Uredia appearing 10 to 14 days after inoculation. Host recovering from attack by end of first month.
Susceptible	3	Uredia medium-sized, 10 to 25 per leaflet. Some stem and petiole lesions.	Uredia erumpent, often surrounded by chlorotic areas, necrotic regions absent. Leaf curling and drying not evident until late, after secondary infection rings appear.	Uredia appearing 8 to 10 days after inoculation. Host and parasite develop somewhat symbiotically for a time; by end of first month leaves beginning to curl and turn brown.
Highly susceptible	4	Uredia large, abundant (25 per leaflet), often nearly covering leaf surface. Numerous stem and petiole lesions.	Uredia large and very erumpent, no necrosis or chlorosis surrounding uredia. Leaves curl and dry up rather readily. Many secondary infection rings, if leaf survives.	Yellowish, chlorotic areas appearing 4 to 5 days after inoculation, uredia evident by end of 7 to 8 days. Rust progresses rapidly and kills leaf early.

After the method of inoculation had been perfected, a varietal-testing program and search for rust-resistant types were begun. From early February to the first week in June, 1940, 2109 rust readings were made in the greenhouse tests. Plants of *Medicago ruthenica* Trantv., *M. falcata* L. and 3 different variegated alfalfas were used in this study, as well as several varieties, introductions, or selections of common alfalfa (*M. sativa*).

Some method of taking accurate notes on infected plants was necessary before a comparison of the susceptibility of varieties could be made. A 0–4 scale was adopted in order to place the rust readings on a numerical basis. The readings were made on 3 general criteria, *i.e.*, coefficient of severity, type of infection, and length of infection period. Each plant examined was given a reading under all 3 criteria, each being based upon the same 0–4 scale. The coefficient of severity denoted the degree to which the plants were infected. Type of infection corresponded to the response of the plants to rust infection, *i.e.*, the general effect of the disease on plants as manifested by chlorosis, "erumpence," etc. The length of infection period concerned mainly the time element in relation to infection. Table 1 shows the infection types and further explains the method used in recording readings on susceptibility.

The total coefficient of infection was determined by finding the sum of the figures for the 3 criteria of rust infection. Dividing the total coefficient by the total number of readings gave a figure that denoted the average coefficient of infection, or the so-called rust reading of the plant. The average of all the readings taken for the plants of a given species, variety, or selection gave the rust reading for that particular species, variety or selection. This method is believed to give an accurate measure of plant and varietal susceptibility.

TABLE 2.—*Summary showing relative susceptibility of species, varieties, and selections of alfalfa to Uromyces striatus under greenhouse conditions, Manhattan, Kans., 1940*

Species, variety or selection	Severity rank	Total plants	Total readings	Coefficient of infection	
				Total	Average
Medicago ruthenica	1	10	30	0.0	0.00
Semipalatinsk	2	24	72	58.5	0.81
Ladak	3	63	189	307.0	1.63
Medicago falcata	4	25	75	128.5	1.71
Kansas Common	5	25	75	181.5	2.42
Kansas Common 1–2018	6	74	222	547.5	2.48
Kansas Common 1–205	7	44	132	343.0	2.60
Grimm	8	77	231	210.0	2.75
Hardistan	9	10	30	97.5	3.25
Turkestan 86696	10	158	474	1552.5	3.28
Turkestan 19304	11	168	504	1681.0	3.34
Hairy Peruvian	12	25	75	267.0	3.57

A summary of the data obtained in the greenhouse (Table 2) shows the comparative resistance to rust of 12 varieties and species of alfalfa. Thus,

the greenhouse tests showed the 16-chromosome species, *Medicago ruthenica*, with a 0.00 reading, to be the most resistant of all species and varieties tested. A strain of Semipalatinsk, *Medicago falcata*, which has crossed naturally with some common alfalfas since its importation from Russia, was given a rust reading of 0.81 and showed a very high degree of resistance in nearly all plants. Ladak proved to be the most resistant of any of the agronomically important varieties. *Medicago falcata* reacted variably. The results of all rust tests indicated that Kansas Common, as a variety, had only a moderate degree of rust resistance (Fig. 2), being less resistant than

FIG. 2. Small uredia of *Uromyces striatus* on the upper surface of greenhouse-grown plants of Kansas Common alfalfa showing chlorotic areas surrounding uredia denoting moderate resistance.

Ladak but considerably more resistant than Turkestan. Grimm ranked eighth among the species, varieties, and selections tested in the greenhouse, rating just below the Kansas Common strains. Turkestan types, including Hardistan, proved uniformly susceptible to rust (Fig. 3). Turkestan 86696 ranked next to Hardistan in the greenhouse tests, with a rust reading of 3.28; it was closely followed by another selection, Turkestan 19304, which had an average coefficient of infection of 3.34. Hairy Peruvian was the most susceptible variety tested in the greenhouse. Rust seemed to make its appearance on the plants earlier than on any of the other species or varieties and also caused more injury.

The wide range in susceptibility among species and varieties of alfalfa was impressive, extending from a reading of 0.00 for *Medicago ruthenica* to 3.57 for Hairy Peruvian. The results with *M. ruthenica* might indicate that plants with a low chromosome count carry a much higher degree of resis-

tance than those with higher chromosome numbers. Further breeding and testing are needed to clarify this point.

Notes on the severity of infection were taken in the field at different dates on 6 varieties or selections of cultivated alfalfa placed in a randomized 2-row series replicated 3 times. Plants were examined carefully for rust appearing on leaves, petioles, and stems (Fig. 4). Each row then was given a numerical rust value from 0–9, following the system of numbering used by the United States Department of Agriculture in reporting damage by

FIG. 3. Large uredia of *Uromyces striatus* on the lower surface of leaflets of susceptible Turkestan alfalfa grown in the greenhouse.

the leaf-spot diseases of alfalfa. A summary of the field reactions of these varieties to rust is presented in table 3. Additional field notes were made on various varieties and selections at the Agronomy Farm plots.

With the exception of the variety, Hairy Peruvian, field and laboratory data seemed, in general, to agree with readings made on the species, varieties, and selections in the greenhouse. Table 4 was prepared with the object of bringing all readings together, so that results could be compared directly. Here, the rankings of the various strains under each of the 3 conditions are shown. The last column in table 4 represents the final rank of each strain

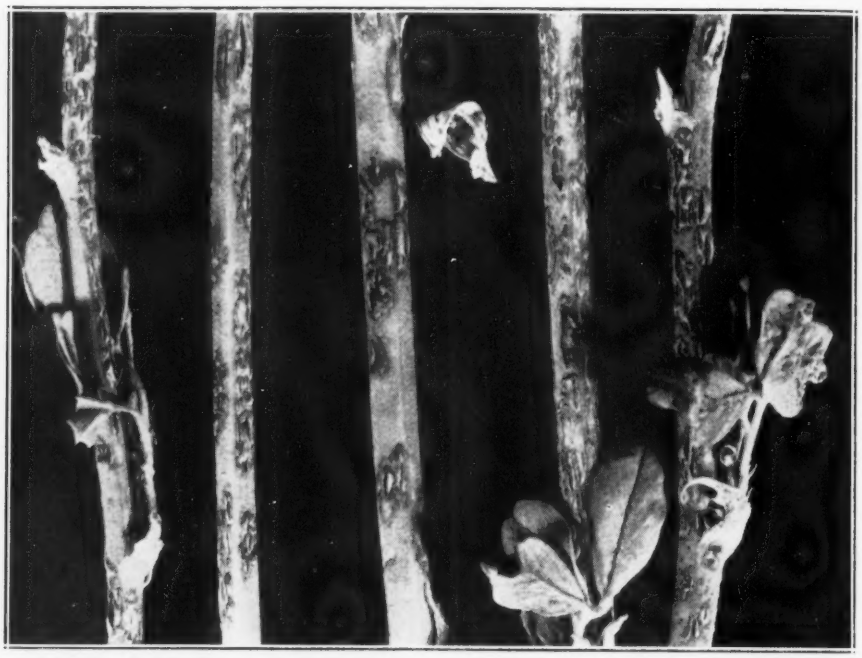

FIG. 4. Large uredia of *Uromyces striatus* on the stems of susceptible Turkestan alfalfa grown in the field.

and indicates its relative susceptibility. Table 4, therefore, shows the rank of each species, variety, or selection for each type of test and its average severity rank for the 3 tests. On the basis of the data shown in this table, *Medicago ruthenica* was the most resistant of all plants tested. Next in rank were *M. falcata* and Semipalatinsk, both of which showed considerable resistance. Ladak ranked fourth, but it might be classed as first among the commercial varieties. Kansas Common types showed only moderate resistance in all cases, hence they all had intermediate ranks. Although Hairy Peruvian proved to be the most susceptible in greenhouse tests, the final rank was somewhat above Grimm and Turkestan. This was due to its tendency to be fairly free from rust in the field during the summer of 1939. Hardistan and Turkestan were by far the most susceptible of any plants tested under each of the three conditions.

TABLE 3.—*Summary of field reactions of alfalfa varieties and selections to rust, Manhattan, Kansas, September–October, 1939*

Variety	Total rows[a]	Total readings	Total severity	Average severity	Severity rank
Grimm	10	3	54.0	3.6	4
Ladak	10	3	39.5	2.6	1
Kansas Common 1–205	10	3	46.0	3.1	3
Kansas Common 1–2018	10	3	43.5	2.9	2
Turkestan 86696	10	3	93.5	6.2	5
Turkestan 19304	10	3	96.5	6.4	6

[a] Rows replicated, readings based on observations on two rows.

TABLE 4.—*Summary table showing relative susceptibility of alfalfa species, varieties, and selections to rust as determined by experiments conducted in the laboratory, greenhouse, and field. Manhattan, Kans., 1939–1940*

Species variety, or selection	Severity rank in greenhouse	Severity rank in field		Severity rank in laboratory		Total tests	Rank in each test on basis of twelve alfalfas tested					Total	Average rank	Final severity rank
		Test I	Test II	Test I	Test II		Green house	Field		Laboratory				
								I	II	I	II			
Medicago ruthenica	1		1		1–2	3	1.0		1.09		1.8	3.89	1.30	1
Semipalatinsk	2		5		3	3	2.0		5.45		3.6	11.05	3.68	3
Ladak	3	1	6	1	5	5	3.0	2.0	6.54	2.0	6.0	19.54	3.91	4
Medicago falcata	4		2–3		1–2	3	4.0		2.73		1.8	8.53	2.84	2
Kansas Common	5		7		4	3	5.0		7.63		4.8	17.43	5.81	6
Kansas Common 1–2018	6	2	2–3			3	6.0	4.0	2.73			12.73	4.24	5
Kansas Common 1–205	7	3		3–4		3	7.0	6.0		7.0		20.00	6.67	7
Grimm	8	4	8	3–4	8	5	8.0	8.0	8.72	7.0	9.6	41.32	8.26	10
Hardistan	9					1	9.0					9.00	9.00	11
Turkestan 86696	10	5	11	5–6	9	5	10.0	10.0	11.99	11.0	10.8	53.79	10.76	12
Turkestan 19304	11	6	10	5–6	10	5	11.0	12.0	10.90	11.0	12.0	56.90	11.38	13
Hairy Peruvian	12		4		6	3	12.0		4.36		7.2	23.56	7.85	9
Kansas 308			9	2	7	3			9.81	4.0	8.4	22.21	7.40	8

The differences in susceptibility in the field afforded an opportunity to select a promising plant of Ladak alfalfa in the plots during the fall of 1939. It was grown in the greenhouse where it was subjected to the most severe rust tests. All efforts to produce rust on the plant failed, hence, it obviously carries a high form of resistance. Cuttings were made from the parent stock, and these also proved resistant. Tests of the cuttings with blackstem, *Ascochyta imperfecta* Peck, made in cooperation with the Department of Agronomy, proved that this rust-resistant selection also is highly resistant to the blackstem disease. Therefore, it seems probable that this selection carries strong resistance to several leaf and stem diseases of alfalfa and should be valuable to alfalfa breeders desiring to use resistant parental stock in crosses. If the resistance carried by this selection could be combined with the good agronomic qualities of Turkestan, a superior strain of alfalfa might be developed for the western Mississippi valley.

SUMMARY

A general study of the relative resistance of different Medicago species and varieties and selections of *Medicago sativa* L. to alfalfa rust (*Uromyces striatus* Schroet.) was made under laboratory, field, and greenhouse conditions.

A method of inoculating excised alfalfa leaves in Petri dishes was developed in the laboratory, which was found to be sufficiently accurate to determine varietal and species differences in susceptibility.

In greenhouse experiments a simple technique was developed by which rust infection comparable to that found in the field was obtained. Fresh rust inoculum was sprayed on potted plants in a moist chamber. Rust pustules appeared 7 to 10 days after inoculation, and final rust readings generally were taken about 3 weeks later.

Marked differences in resistance to rust among varieties and species of Medicago were observed in the greenhouse. *Medicago ruthenica* carried the highest form of resistance. Turkestan and Hairy Peruvian were the most susceptible. With the exception of Hairy Peruvian, readings on species, varieties, and selections made in the greenhouse and laboratory agreed very closely with those taken in the field. This one variety appeared susceptible in the greenhouse but was relatively free from rust in the field during the summer of 1939.

A plant selection of Ladak made in the field exhibited the most resistance of any strains or varieties of the *Medicago sativa* group. This selection not only showed high resistance to rust in greenhouse tests but also recently exhibited high resistance to blackstem disease of alfalfa caused by *Ascochyta imperfecta*. Cuttings of this selection have been released to the alfalfa improvement project of the Kansas Agricultural Experiment Station, Manhattan, Kansas, for other tests and possible increase.

KANSAS AGRICULTURAL EXPERIMENT STATION,
MANHATTAN, KANSAS.

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QUANTITATIVE MEASUREMENT OF A STRAIN OF TOBACCO-ETCH VIRUS

FRANCIS O. HOLMES

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More than a dozen phytopathogenic viruses can now be measured by noting the incidence of conspicuous primary lesions on inoculated leaves of appropriate host plants. These include the viruses of tobacco mosaic (7), potato mottle (17), tobacco ringspot (13), tomato spotted wilt (18), cucumber mosaic (14), alfalfa mosaic (21), tobacco necrosis (20), turnip mosaic (6), tomato ringspot (15), tobacco streak (12), tobacco Bergerac ringspot (19), tomato bushy stunt (2), potato yellow dwarf (3), pea streak (4), and pea wilt (11).

The primary-lesion method for measurement of virus activity is considerably more efficient than the most widely applicable alternative technique, which depends on the percentages of inoculated plants that become diseased at specific dilutions of inocula. In the primary-lesion method, each inoculated leaf is capable of disclosing a number of separate infections or their absence, and thus serves in the same capacity as a set of plants in the alternative method. The relative facility with which hitherto obscure properties of viruses can be investigated when the primary-lesion method becomes applicable has led the writer to hope that many, if not all, mechanically inoculable viruses eventually might be susceptible to study by this technique if further information on their potential host ranges should become available. The results from experiments with tobacco-etch virus tend to support this view.

Tobacco-etch virus (*Marmor erodens* H.) was selected for further study, several years ago, because it was a conveniently available representative of viruses then known to be readily transmitted by mechanical means from one host to another, but not known to be capable of inducing formation of conspicuous primary lesions in any susceptible species. A search was begun for a host in which it would produce conspicuous primary lesions. The severe-etch strain (var. *severum* H.) was used, because it seemed likely to induce more severe and conspicuous manifestations of disease than the type strain (var. *vulgare* H.).

The severe strain of tobacco-etch virus was originally studied and described by Johnson (10). It induces in Turkish tobacco and other varieties of *Nicotiana tabacum* L. a disease that is characterized both by chlorotic mottling and by intricate patterns of fine white lines that appear as though etched on the surface of affected leaves; in most other known hosts it induces chlorotic mottling without etched patterns.

Primary lesions are rarely discernible in hosts infected by the severe-etch strain. They are, however, sometimes to be seen in inoculated leaves of the most commonly studied host, tobacco. There they appear as faintly chlo-

rotic spots, as fine, white, etched rings, or as combinations of the two. These primary lesions have been made clearly visible by Bawden and Kassanis (1) through staining infected tobacco leaves with iodine; the lesions, in which there is abnormal retention of starch, then can be counted accurately. In living leaves, they are not conspicuous enough to be counted readily in this or any other host that was known before the present investigation was begun.

In the search for a useful test plant for quantitative estimation of tobacco-etch virus, more than 250 species of plants were inoculated. About one-fourth of all the tested species permitted increase of the virus in their tissues; a majority of these also showed disease manifestations. Among the species that were found susceptible, a few, but only a few, showed necrotic primary lesions as a result of infection. Of these, *Physalis peruviana* L., the Cape gooseberry, proved best for estimating virus activity, as has already been reported in an abstract (9).

The characteristics of primary and secondary lesions in *Physalis peruviana*, the influence on them of age and condition of host, season, manner of inoculation, and source of virus, as well as results of some measurements made by use of the primary lesions, form the subject of the present paper.

PRIMARY AND SECONDARY LESIONS IN PHYSALIS PERUVIANA

Primary Lesions. Inoculation of *Physalis peruviana* with the severe-etch strain of tobacco-etch virus was followed in about 5 days, under appropriate conditions, by collapse of tissues in little spots on the inoculated leaves. The spots, which constituted necrotic primary lesions, later became larger and displayed a dark brown zone of pigmentation surrounding a tan or light brown center; outside these two necrotic zones there was often a third, less well defined zone of yellowing. The lesions became increasingly conspicuous until about the tenth day after inoculation, when final counts for quantitative studies usually were made. Typical necrotic primary lesions in a partly yellowed, inoculated leaf of *P. peruviana*, 10 days after inoculation, are represented in figure 1.

Old yellowing leaves sometimes showed primary lesions as chlorophyll-retention patterns without accompanying necrosis. On other occasions they showed them as spots of chlorophyll retention with but small central areas of necrosis. In either case, if lesions were numerous, these old leaves often dropped before younger leaves were ready for final counting. Because of this, records were kept from the 6th or 7th day after inoculation until about the 10th day.

Even relatively young leaves that developed many lesions tended to become yellow and to abscise earlier than comparable healthy leaves or leaves that showed fewer lesions. This abscission of inoculated leaves, though in a way comparable to the loss of inoculated leaves of the Tabasco pepper and its derivatives when inoculated with tobacco-mosaic virus (8), differed in that it did not occur promptly enough to prevent systemic spread of virus.

Secondary Lesions. Systemic spread of the virus often induced the ap-

pearance of secondary lesions in young developing leaves about the 10th day after inoculation, sometimes earlier. The secondary lesions, when they were so few as to develop without interfering with each other, resembled the primary lesions in general appearance. In young leaves that were heavily

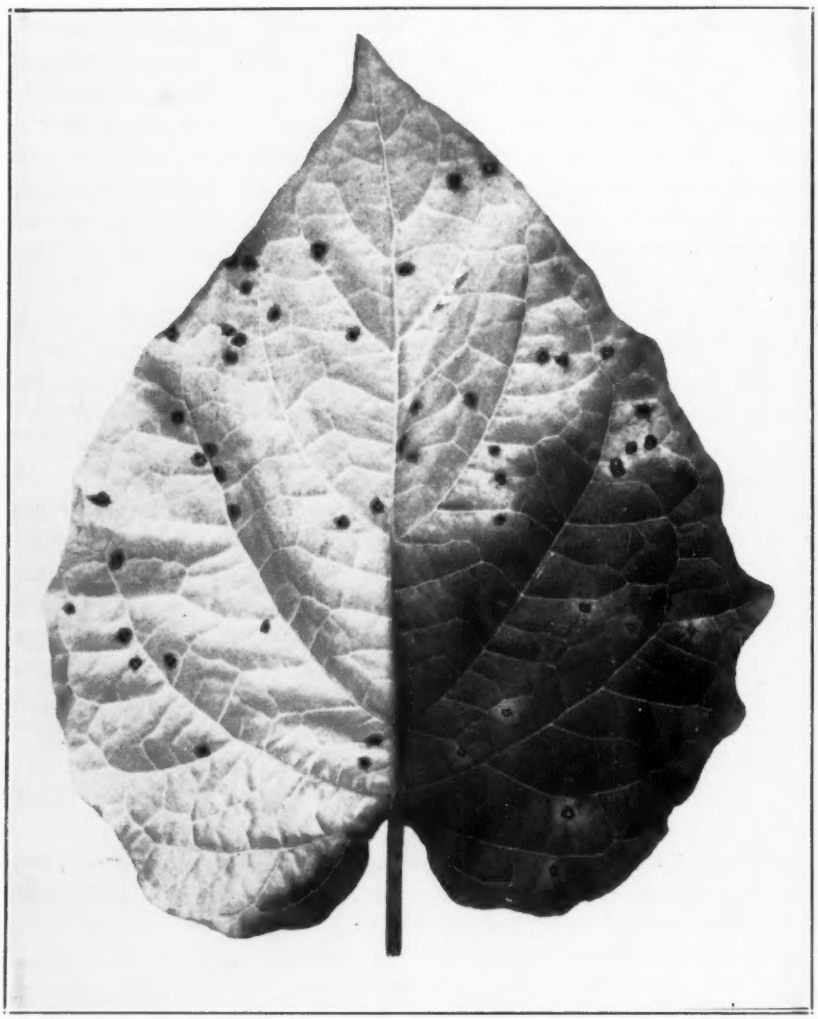

FIG. 1. Primary lesions in an inoculated leaf of Cape gooseberry, *Physalis peruviana*, 10 days after inoculation with the severe-etch strain of tobacco-etch virus. Lesions necrotic, dark brown peripherally, with lighter brown centers; in the lower right portion of the leaf, which had not yet turned yellow at the time of photographing, the green lamina was set off from each lesion by a diffuse yellow halo.

invaded, abscission sometimes occurred too early to permit development of recognizable, discrete, necrotic lesions; wilting, however, sometimes occurred in these young leaves just before they fell from their stems.

Factors Affecting the Use of Primary Lesions in Measurement of Virus

Concentrations. The type of primary lesion represented in figure 1 is perhaps the most desirable for quantitative measurement studies. It presents considerable necrosis, little or no confluence, conspicuous yellow peripheral halos in green areas of leaf tissue, high contrast in color in yellowed areas. Deviations from this type of lesion were found to occur in consequence of a number of variables that must be recognized in practical work.

Age of Test Plant. Plants proved best for use in virus estimation about the time of first flowering. Younger plants were affected so promptly by systemic necrosis as to be killed outright in a more or less diagnostic manner. They showed, in quick succession, abscission of leaves just below the top of the plant, abscission of top leaves and of the older or of all inoculated leaves, death of stem tips, and eventual collapse of the whole plant. Often the

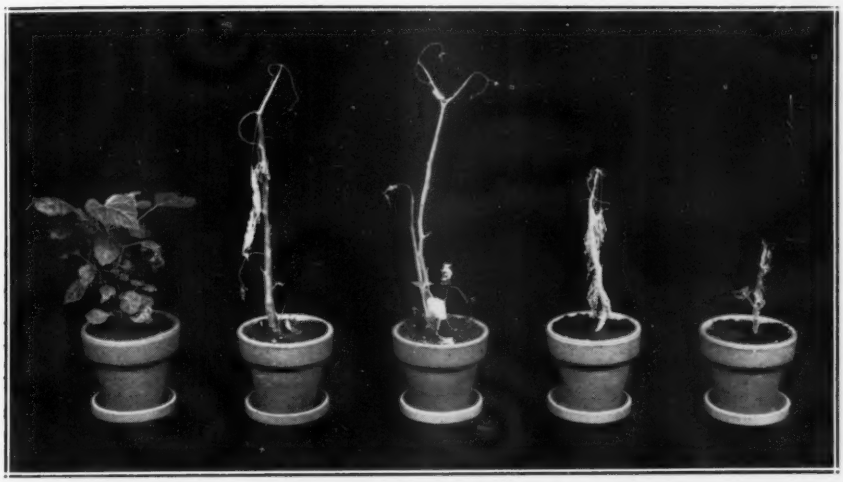

FIG. 2. Specificity of protection against the severe-etch strain of tobacco-etch virus in plants of *Physalis peruviana* inoculated with (left to right) the mild-etch strain of the same virus, healthy tobacco juice, cucumber-mosaic virus, tobacco-mosaic virus, and the Canada-streak strain of potato aucuba-mosaic virus. Only the plant at the extreme left escaped systemic necrosis as a result of the test inoculation.

whole succession of events was so rapid that it was concluded without formation of discrete primary lesions.

Age of Inoculated Leaf. In general, plants proved less susceptible in their younger than in most of their older leaves; on some plants the very old leaves were also relatively insusceptible.

Seasonal Effects. Fall, winter, and spring conditions in the greenhouse, with temperatures held as close as possible, at least at night, to 22° C., proved satisfactory for making quantitative measurements. Summer conditions were unsatisfactory; the trouble appeared to involve unknown factors other than temperature, probably acting before inoculation. Leaves of *Physalis peruviana* grown under summer conditions were thicker and tougher than those grown in fall, winter, or spring and permitted only a few, highly localized lesions to form as a result of inoculation; natural immunity seemed to be approximated under these conditions.

Effect of Changing Strain of Virus. Conspicuous, necrotic, primary lesions like those characteristic of the severe-etch strain did not occur as a result of inoculations made with the mild-etch strain, the only other strain of tobacco-etch virus that was tested. The mild-etch strain generally induced a systemic, chlorotic-mottling disease in *Physalis peruviana*. Only occasionally some necrosis, local or systemic, was observed. Definite relationship of the two strains was shown, however, by the fact that chlorotic areas of the plants infected by the mild-etch strain showed no evidence of being infected after reinoculation with the severe-etch strain. The second inoculation produced neither necrotic primary lesions nor systemic necrosis. Similar protection was not conferred by preliminary infection with other viruses that caused chlorotic mottling in the same host, such as cucumber-mosaic virus (*Marmor cucumeris* H.), tobacco-mosaic virus (*M. tabaci* H.), or Canada-streak virus (*M. aucuba* H. var. *canadense* Black and Price). Figure 2 shows the specificity of protective inoculation with mild-etch virus. The findings confirmed the observation of Bawden and Kassanis (1) that protection against severe-etch virus is afforded by prior infection with mild-etch virus.

Effects of Intentional Variations in Manner of Making Test Inoculations. Several modifications of technique were tested to discover the best manner of conducting inoculation experiments. As a standard practice, plants of *Physalis peruviana* growing in 6-inch clay pots were inoculated, about the time of blossoming or first fruiting, by rubbing the upper surface of each large leaf with a muslin pad moistened liberally with the infectious fluid to be tested. Usually, 10 or more leaves of each plant were inoculated, and the 6 successive leaves that proved most highly susceptible were selected for counting.

Increasing the number of strokes for the inoculation of each leaf gave a slight increase followed by a decrease in primary lesions; thus with 6, 12, 18, and 24 strokes per leaf, the lesion counts per set of 12 test leaves (6 on each of 2 plants) were 1633, 1714, 2250 and 1642, respectively. Perhaps 12 strokes per leaf should be recommended as conserving time and producing numbers of lesions not likely to be greatly modified by slight variations in length or severity of strokes.

Rinsing leaves with water immediately after inoculation, 15 minutes later, or not at all, did not greatly modify results, giving averages of 38.8, 50.9, and 53.3 lesions per leaf, respectively, in a test that involved 36 inoculated leaves in each of the 3 categories. Less favorable environmental conditions or chemical constituents of inocula other than those thus far tested may, nevertheless, justify rinsing plants after inoculation.

Removing the growing tips of test plants did not appreciably affect the gradient in numbers of lesions on successive leaves; it tended to induce early appearance of secondary lesions and was discontinued.

Reducing the supply of fluid on inoculating pads, or continuing the use of pads without remoistening, resulted in unexpectedly early and progres-

sive exhaustion of infectivity. Liberal renewal of inoculum proved essential for obtaining uniform results from successive inoculations. A freshly moistened or newly remoistened pad of cloth was generally used for inoculating each test plant, and in some experiments the inoculating pad was remoistened after inoculating each pair of successive leaves with even better results than were obtained when it was remoistened for each plant.

USE OF PHYSALIS PERUVIANA IN STUDYING SOME PROPERTIES OF THE VIRUS

Various Species as Sources of Virus. By the use of *Physalis peruviana* to measure virus activity, production of the virus by 5 common host plants was compared quantitatively, with a view to choosing a satisfactory source of virus for future experiments. Virus production in inoculated leaves 10 days after inoculation proved greatest in *Nicotiana glutinosa* L., successively less in *N. tabacum, Lycopersicon esculentum* Mill., *Datura stramonium* L., and *Capsicum frutescens* L. The degree to which these plants differed as virus sources in this comparison of their unmodified expressed juices may be judged from the respective counts of 1683, 698, 422, 6, and 5 lesions per set of 6 test leaves. Juices from *D. stramonium* proved more infectious when diluted 1:10 with water than when not diluted; therefore, the apparently low virus content of this host may be the result of a toxic effect of its juices on the test plant rather than an effect of slow or limited production of virus within its tissues. It was in this species, *D. stramonium,* that a strain of tobacco-etch virus was first recognized in nature, by Fernow (5) who called it the virus of mosaic C.

Many experiments were performed before the relative value of various species for virus production had been established. *Nicotiana tabacum,* which proved to be second in virus productivity, was used as source of virus for all of these experiments. Some later studies requiring high titer were made with material from *N. glutinosa,* however.

Rate of Increase in Tobacco. Measurement of virus from inoculated tobacco leaves and from the tops of corresponding plants at various intervals showed that inoculated leaves were, on the whole, better sources of virus than systemically infected top leaves. Activity reached a maximum in samples from both locations about 10 days after the plants' initial inoculation and then fell off, more rapidly in the systemically diseased tops than in inoculated leaves. The early increase of activity and the subsequent decline are shown graphically in figure 3. The phenomenon is comparable to that found by Ross (16) in the study of alfalfa-mosaic virus (*Marmor medicaginis* H.).

Effect of Drying. Inoculated leaves of insusceptible species of plants proved consistently free of virus when tested after 10 days, even though undiluted expressed juices from diseased plants had been applied to them originally. This led to the supposition that the virus might not survive long when dried. To test this point, juices were expressed from top leaves of

plants of *Nicotiana glutinosa* 10 days after inoculation of lower leaves of the same plants. These juices were at once exposed to the air of the laboratory in 1 cc. amounts in shallow glass dishes. They became dry within an hour. Tests of infectivity were made at intervals of 0, 1, 2, 3, 5, 7, and 10 days by resuspending the air-dried residues in the original volume of water and using them as inocula for plants of *Physalis peruviana*. The results showed that complete inactivation was by no means attained within 10 days, lesion counts from 12-leaf inoculations made at the specified times being 4563, 1656,

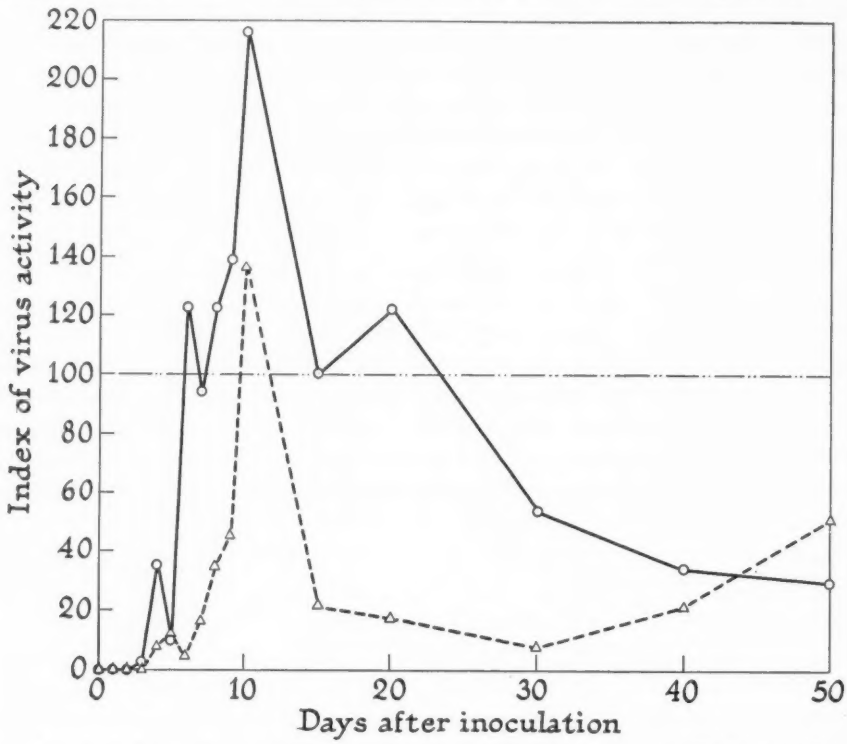

FIG. 3. Infectivity of juices expressed from inoculated leaves (solid line) and top leaves (dotted line) of tobacco plants at intervals after inoculation with the severe-etch strain of tobacco-etch virus. The index that is represented expresses a quotient: the number of lesions on a plant of *Physalis peruviana* inoculated with the indicated sample divided by the number of lesions on a control plant inoculated on each occasion with juice from a tobacco leaf infected 10 days earlier.

736, 578, 519, 304, and 121, respectively. A control test of freshly expressed juice made on the 10th day gave a corresponding lesion count of 2137, lower than the count indicating original infectivity but higher than any subsequent measurement in the series. The lower value of the control perhaps reflects in part the difficulty of duplicating a sample of infective juice at or near maximum infectivity, and in part a gradual decrease in susceptibility of a set of test plants with age. The experiment shows that infectivity of expressed juice is gradually decreased by drying, but that a substantial infectivity still remains after a lapse of at least 10 days, a period in which the

virus might, under some circumstances, be transported intentionally or unintentionally over considerable distances.

It became of interest to test whether virus would survive equally well within dried tissues of diseased leaves. Similar source leaves were dried, therefore, without being crushed. The drying process required more time than had been needed for small juice samples. Leaves were leathery in texture and brittle in parts after 1 day of drying in the air of the laboratory, brittle throughout after 2 days, but continued to lose weight even after the 3rd day; by the 5th day they had reached a weight that was subsequently maintained. The final air-dry weight amounted to 10.0 per cent of the original green weight. Tests of infectivity were made by remoistening and grinding the drying leaves at the intervals used in the dried-juice experiment. Lesion counts from 12-leaf inoculations in this series were somewhat lower: 3174, 122, 3, 0, 38, 31, and 9, respectively, with a corresponding control count of 397 from a fresh-leaf sample tested on the 10th day of the experiment. Again it appeared that considerable reduction of activity was experienced during a 10-day period of drying, but that dried diseased leaves remained capable of furnishing some virus for this length of time at least.

Effect of Storage at Low Temperatures. Juice samples, diluted 1:10 and stored at low temperatures in the presence of acid buffer solutions (pH range between 4.5 and 6), retained infectivity well. A sample giving an original 6-leaf lesion count of 72 still was capable, after 10 days just above freezing temperature in a refrigerator, of giving a count of 43, and, after a like period just below freezing temperature in a cold-storage room, of giving a count of 51. Both in the chilled and in the frozen sample sedimentation

TABLE 1.—*Inactivation of severe-etch virus by heat. Numbers indicate lesions produced on single plants of Physalis peruviana inoculated with samples of undiluted juices from severe-etch tobacco plants, after the samples had been heated at various temperatures and for the indicated intervals of time*

Heating period	51° C.	53° C.	55° C.	57° C.	59° C.
0 min.	237	436	402	311	417
5 "	31	14	2	0	0
10 "	30	6	0	0	0
15 "	27	2	0	0	0
30 "	18	0	0	0	0

occurred, leaving a clear supernatant fluid of very low virus content; almost all the surviving virus seemed to be associated with the sedimented fraction.

Effect of Dilution. Upon water dilution of expressed juice from diseased plants of *Nicotiana glutinosa*, infectivity for *Physalis peruviana* was progressively decreased. Dilutions proved weakly infective at 1:1000, uninfective at 1:10,000. Successive dilution of 1:1, 1:2, 1:10, 1:100, 1:1000, and 1:10,000 gave lesion counts of 277, 98, 47, 7, 0.7, and 0 per set of 6 test leaves. Had a more concentrated original sample been used, the apparent dilution end-point might have been somewhat different from that found in this experiment.

Thermal Inactivation of Virus. Undiluted juice from severe-etch tobacco plants remained infective after exposure for 10 minutes at a temperature of 53° C., but was completely inactivated by exposure for the same length of time at 55° C. When the time of heat treatment was extended to 30 minutes, some infectivity could be demonstrated at 51° C., though not at 53° C. nor at any higher temperature that was tested. These and other details of the results of heat treatments are shown in table 1.

SUMMARY

Conspicuous necrotic primary lesions were produced in leaves of *Physalis peruviana*, under some environmental conditions, 5 to 10 days after inoculation with the severe-etch strain of tobacco-etch virus. Quantitative measurements of this virus could be made by using the number of lesions resulting from each inoculation as an index of the infectivity of the inoculum.

By the use of this method of measuring the virus, several plant hosts were compared as potential virus sources; *Nicotiana glutinosa* appeared to be the best among them. *N. tabacum* and *Lycopersicon esculentum* furnished less infective extracts but were fairly satisfactory sources 10 days after initial inoculation. Virus activity in expressed juices of *N. tabacum* reached a maximum about 10 days after inoculation and then declined rapidly.

Severe-etch virus was found capable of withstanding drying for at least 10 days in juice samples and in diseased leaves, although in both cases there was a reduction of infectivity with increased time. It retained activity well for at least 10 days in acid-buffer solutions (pH 4.5 to 6) at temperatures just above and just below freezing. It retained some infectivity after being heated for 10 minutes at 53° C., but was completely inactivated in this length of time at 55° C.; it was still viable to some extent after 30 minutes at 51° C., but was wholly inactivated when exposed to a temperature of 53° C. for the same period.

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BLUEBERRY CANE CANKER

J. B. DEMAREE AND MARGUERITE S. WILCOX[1]

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A canker disease of stems and shoots of blueberries (*Vaccinium* spp.) was first observed[2] in a North Carolina cultivated field in 1938. Later surveys showed the disease widely distributed in cultivated fields of the highbush swamp blueberry (*V. australe* Small) in North Carolina and Georgia and in cultivated rabbit-eye blueberry (*V. ashei* Reed) in Alabama, Florida, and Mississippi. It was also found in wild *V. australe* in North Carolina and in wild growth of *V. ashei* in west Florida.

The disease is caused by a fungus of the *Physalospora* type that produces both perithecia and 2 forms of pycnidia (macro and micro) in infected cortex tissues. It is probably indigenous to southeastern United States, as it has been observed in wild blueberry bushes growing not only adjacent to cultivated blueberry fields but in localities several miles distant from plantings. Cankers have been seen in wild bushes in North Carolina of size and character that would indicate infection antedating the establishment of the pioneer cultivated fields in the State. The older blueberry fields in North Carolina, nearly all of which now show more or less canker, were planted with nursery stock grown in New Jersey, a section where the canker is unknown; therefore, it is evident that the disease, so prevalent in North Carolina, has spread to cultivated fields from infection foci in wild bushes.

The rabbit-eye blueberry industry in west Florida was established 30 years or more ago from seedling plants taken from wild growth along river courses of Florida and Alabama, and probably diseased plants were introduced into cultivated plantings at that time. Some canker is now present in most of those cultivated fields, varying from an occasional infected bush in some fields to as much as 50 per cent infection in others. All degrees of susceptibility, from near-immunity to complete susceptibility, are manifest in those seedling plantings. The primary shoots of very susceptible bushes show almost solid canker development from the ground line to a height of 3 or 4 feet. In spite of the extensive cankers, no substantial mortality of bushes has occurred, although many become weakened and make poor growth.

The pathogen gains entrance through the unbroken bark, probably through lenticels of current-year shoots. The points of entrance become evident in late summer or early fall as reddish, broadly conical swellings. Pycnidia usually develop on the surface of these conical growths and the bark may or may not crack the first season (Fig. 1, A). Later symptoms

[1] The writers are indebted to Dr. W. W. Diehl for assistance in identifying the fungus, to Miss Edith Cash for the Latin translation of the technical diagnosis, and to Mr. G. A. Meckstroth for certain observations and for numerous specimens.

[2] The disease was called to the attention of the writers by Mr. C. A. Doehlert, New Jersey Agricultural Experiment Station, who collected specimens near Magnolia, North Carolina, in December, 1938.

vary considerably, depending upon the host variety involved. The second year after infections occur on the varieties Cabot and Pioneer the small swellings lose their reddish color and conical shape, enlarge somewhat, and become fissured. The fungus invades the cortex tissues in all directions from points of infection and this newly invaded bark tissue swells, the surface becomes uneven, resembling blisters, and turns light gray (Fig. 1, B). The fungus not only invades the cortex and cambium but also penetrates slightly into the xylem, causing some discoloration but no extensive necrosis

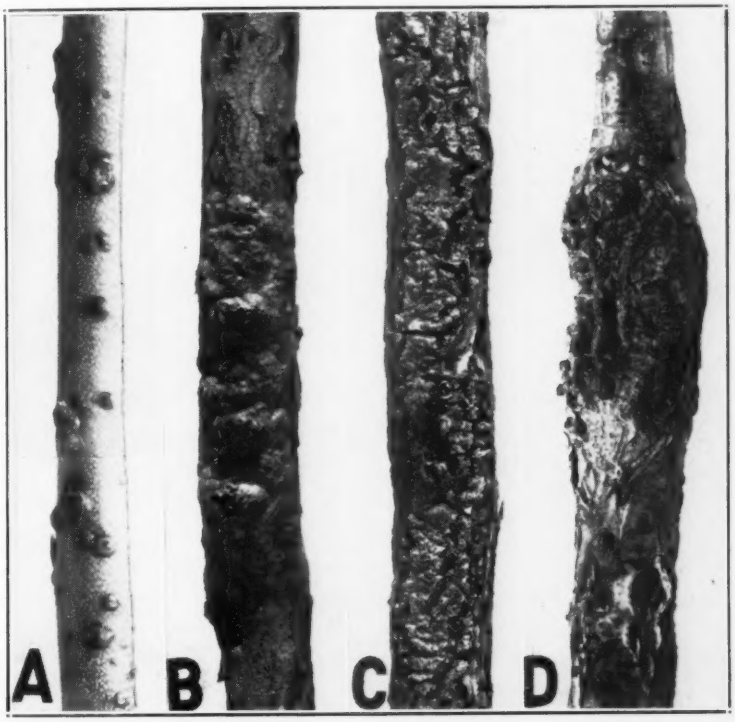

FIG. 1. Blueberry cane canker caused by *Physalospora corticis*. A. Early stage of canker infection on current year's wood. ×1½. B. Active canker about 3 years old showing "blister stage." The surface of the bark is uneven and grayish and fissures have begun to appear. ×1½. C. An old, deeply fissured canker. ×1. D. Typical callus formation around a shoot wound artificially inoculated with the canker fungus. ×2.

of the wood. By the end of the second year or the beginning of the third the older portions of the cankers are rough, black, and deeply fissured (Fig. 1, C). The result is girdling of the shoots, the parts above becoming unfruitful and finally dying. For some unexplained reason cankers first appear on the side of shoots directly exposed to the sunrays. This feature is well brought out in figure 2, A and B.

The cortex reaction to the fungus in some other blueberry varieties studied is less pronounced than in Cabot and Pioneer. The cankers may not be conspicuous but, instead, may show as only slightly swollen, uneven, gray-

ish areas, with little or no cracking of the bark. The injury to the shoots of such varieties is much less severe than if cracking of the bark occurs.

VARIETAL SUSCEPTIBILITY

Varieties of the high-bush blueberry show considerable difference in their ability to resist attack by the canker fungus. The variety Cabot is doubtless the most susceptible and will perhaps be discarded in the South for this

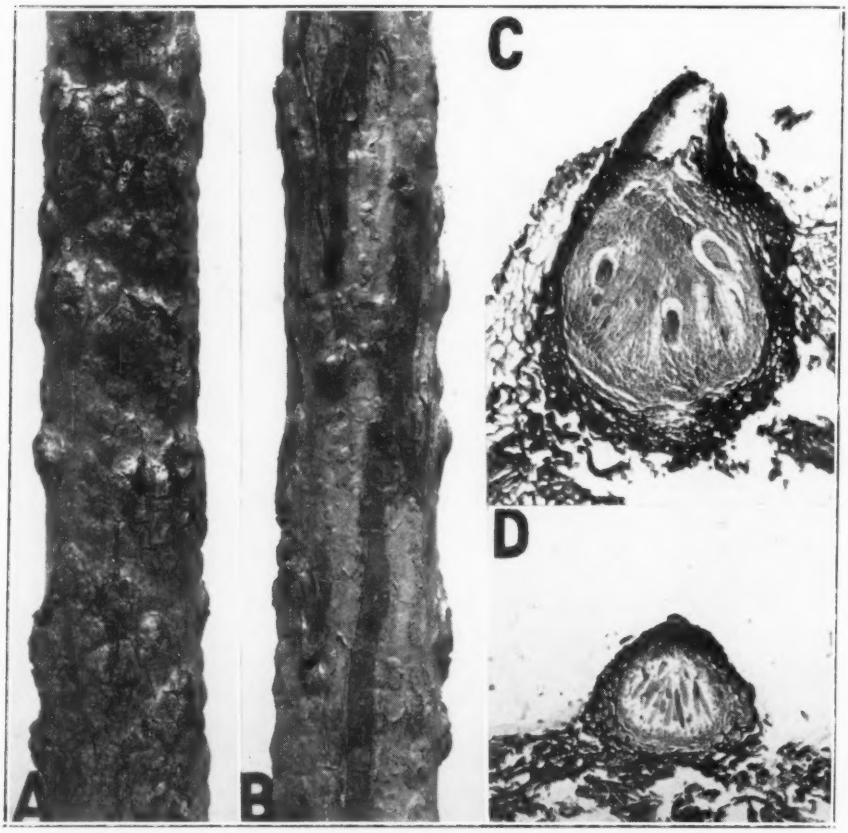

FIG. 2. A. Characteristic appearance of canker on side of a blueberry cane exposed to sunrays. B. Opposite side showing only scattered lesions and blisters. ×1. C. Perithecium of *P. corticis* showing immature asci surrounded by paraphysoid filaments. ×150. D. Pycnidium. ×150.

reason. Other observed varieties, in descending order of apparent susceptibility, are as follows: Pioneer, Concord, June, Stanley, Jersey, Scammell, Rancocas, and Rubel.

Named varieties of the rabbit-eye blueberry are mostly of recent selection and all have not been subjected to severe tests for susceptibility. The varieties Black Giant, Hagood, Locke, Myers, Owens, Ruby, and Scott have been examined for presence of cankers occurring naturally and they were found only on Locke.

THE FUNGUS

Pycnidial Stage

Pycnidia form in the summer, are sometimes abundant in the margin of cankers, and are frequently present on new shoot lesions (Fig. 1, A) near the termination of the first growing season after infection takes place. They are scattered, partly imbedded or superficial, black, conical to subglobose, ostiolate; those measured varied from 90–208 μ high and 60–165 μ wide (Fig. 2, D). The majority were from 105 to 140 μ by 70 to 100 μ. The pycnidial wall is carbonaceous, and rather thick, varying from 10 to 30 μ wide. The pycnospores (Fig. 3, C) are fusiform elliptical, and some are obtuse at the

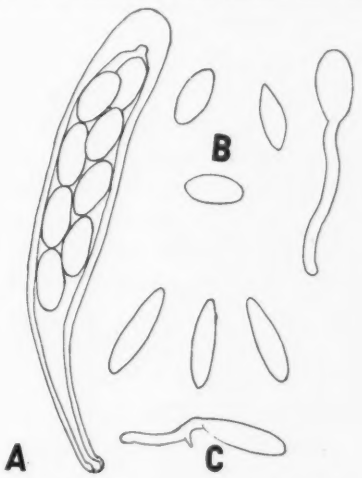

FIG. 3. *Physalospora corticis*. A. Ascus. B. Ascospores. C. Pycnospores. All about × 320.

base and pointed at the opposite end. They are nonseptate, hyaline, averaging 35 μ long and 9 μ wide, and develop from conidiophores about 15 μ long. Another type of spore has occasionally been observed either in pycnidia or in other fruiting bodies somewhat smaller than true pycnidia. These spore structures are rod-shape, hyaline, nonseptate, about 3 to 7 μ long, and are produced on relatively long conidiophores. They are doubtless a stage in the life history of the fungus, since they form when mycelium from subcultures derived from ascospores has been used to inoculate autoclaved corn meal.

Perithecial Stage

The perithecia are produced much more abundantly than pycnidia. Those on the older portion of cankers are usually empty, but functioning ones often can be found in late summer in the more recently invaded bark tissues. They average about 280 μ high and 200 μ wide. The typical shape is definitely conical, and the ostioles of some are distinctly pointed and drawn out into short beaks 50 to 100 μ long. Otherwise, the perithecia have about the same superficial appearance as pycnidia.

Perithecia contain at first white, erect, closely packed, short-celled, anastomosing hyphae (Fig. 2, C). Shear, Stevens, and Wilcox,[3] in discussing the character of *Botrysphaeria ribes* and *Physalospora malorum* (*obtusa*), considered similar filamentous perithecial structures to be a form of paraphyses. The asci develop from the base of the perithecium and push up through the hyphal mass. There are seldom more than a dozen asci in a perithecium, frequently only 4 to 8, separated by layers of paraphyses (Fig. 2, C).

The asci usually are clavate, contain 8 spores, and the wall is much thicker at the apex than elsewhere (Fig. 3, A). Actual liberation of ascospores has not been observed, but it is not unusual in water mounts to find asci ruptured in a characteristic manner. These asci were broken transversely immediately below the thickened cap, and the cap was pushed forward a short distance by elongation of the ascus contents. The spores did not drift apart but retained the same outline of the ascus as though they were held together by a colorless gelatinous substance or a very thin membrane, which elongated below and was attached to the inside base of the ascus.

The ascospores (Fig. 3, B) are hyaline or sometimes slightly tinted, ellipsoid to fusoid, and nonseptate. The size of those measured averaged 29.2 μ long and 11.7 μ wide.

Ascospores have been observed to germinate within 30 minutes after being mounted in tap water. The germ tubes elongated rapidly and formed unbranched hyphae approximately 120 μ long in 5 hours. At the end of a 19-hour observational period the length of the primary hyphae varied from 142 to 380 μ and no branching occurred. The spores frequently germinated while still in unruptured asci, the germ tubes passing through the ascus wall, even through the thick apex, with little or no apparent retardation in rate of growth.

CULTURAL CHARACTERISTICS

On corn-meal agar the blueberry canker fungus grew rapidly, covering the surface of agar in an ordinary Petri dish in 6 or 7 days at a temperature of 25° C. The mycelium was light to dark green, septate, coarse, and sparsely branched. Loose felty hyphae covered the surface of the colonies. No form of fructification has been observed in cultures on corn-meal agar but some pycnidia formed in flasks of autoclaved corn meal.

PROOF OF PATHOGENICITY

Pathogenicity of the *Physalospora* was demonstrated by inoculating potted blueberry plants in a greenhouse with subcultures from single ascospore isolations. The ascospores were taken from typical blueberry cankers collected in North Carolina.

Since spores could not readily be obtained from cultures, bits of agar

[3] Shear, C. L., Neil E. Stevens, and Marguerite S. Wilcox. *Botryosphaeria* and *Physalospora* on currant and apple. Jour. Agr. Res. [U.S] 28: 589–598. 1924.

containing mycelium of the fungus were used as the inoculating material. The inoculum was applied to wounded and nonwounded surfaces of shoots of different ages, then covered with damp cotton and wrapped with parafilm. The wounding was done by making a short longitudinal scratch in the bark. Uninoculated plants, similarly treated, served as controls.

In March and April, 1939, 54 inoculations were made on blueberry seedlings. Wounds were made for 25 of the inoculations, while 29 were not wounded. One- and 2-year-old shoots principally were used. At the termination of the growing season there was neither evidence of infection on any of the nonwounded shoots nor any clear indication of canker formation on the wounded ones. All except 9 of the wounded shoots, however, produced a callus formation around the wound, and a few *Physalospora* pycnidia formed in the callus tissues of some, but no swelling, unevenness, cracking or other signs characteristic of canker, known under field conditions, developed. The presence of callus growth and of pycnidia thereon was not considered a criterion of infection, and the plants were discarded. Plants similarly inoculated in 1940, as described below, were held over for observation through 1941, when it was found that a single growing season was insufficient time for development of definite signs of pathogenicity of the fungus. This suggests that the 1939 inoculated plants may have been discarded before there was time for typical symptoms to appear.

In 1940, the work was repeated in about the same manner as the previous year except that plants of the very susceptible variety Cabot were used rather than seedlings. Thirty-four inoculations were made principally in wounded succulent and 1-year-old wood. Final observations made at the end of the growing season showed results similar to those obtained the previous season, *i.e.*, unwounded plants showed no evidence of infection but all wounded shoots made the same type of callus formation as was noted in 1939.

At the close of the growing season of 1941 the plants inoculated in the spring of 1940 showed a high percentage of canker. There were typical canker signs on all wounded inoculated shoots, of which 11 were of the current season's growth, 15 were 1 year old, and 1 was 2 years old when inoculated. Of 4 nonwounded succulent shoots 1 showed canker development; of 3 nonwounded 1-year-old shoots all showed cankers. At the end of the second year the callus formation on most of the inoculated wounded shoots had enlarged and in a few cases had girdled the shoot (Fig. 1, D). The most positive indication of the pathogenic nature of the organism used for the inoculation was the extension of an infected area around the point of inoculation as shown by culturing the cortex tissues, by the grayish color, unevenness, and cracking of the bark surface, and by the presence of perithecia and pycnidia, all being characters common in newly invaded bark tissues adjacent to naturally-formed cankers. Signs of the fungus invasion extended from 2 to 5 cm. above and below the area where the inoculum was applied.

IDENTITY OF THE FUNGUS

Provided the tangled anastomosing hyphae within the perithecia and among the asci of this blueberry canker fungus can be considered of a paraphysoid nature, its morphological characteristics would then place it in the genus *Physalospora*. Species of the closely related fungus *Botryosphaeria* commonly form perithecia in groups on a stroma. Although some stromatic material is mixed with the blueberry canker tissues, the perithecia are distinctly scattered and do not necessarily grow from stromatic tissue.

The blueberry fungus is similar in some respects to *Physalospora obtusa*. Both fungi have the same type of filamentous packing around the asci. Asci and ascospores are about the same size and shape and are similar otherwise. The apical portions of asci of both are considerably thickened and both exhibit the same peculiar method of rupturing of asci back of the thickened tip. The germ tube of the blueberry fungus elongates to a considerable length before branching, a characteristic also common for *P. obtusa*. Both fungi produce two types of pycnospores—macro and micro. There are, however, clearly some differences between the 2 fungi. The mature macropycnospores of *P. obtusa* are brown, while those of the blueberry fungus are hyaline and of a distinctly different size and shape. When grown in Petri dishes on corn-meal agar, under identical conditions, the apple fungus makes colonies twice the diameter of those of the blueberry fungus. The blueberry *Physalospora* produces perthecia more abundantly than pycnidia, while the pycnidial stage of *P. obtusa* is the more common. Another difference between the 2 fungi is the reaction when they are inoculated into apple fruits. *P. obtusa* produces an extensive and rapid decay in apples followed by the production of *Sphaeropsis* pycnidia. When the blueberry canker fungus was inoculated into apple fruits no decay developed. Therefore, although the 2 fungi are similar in some respects, they are so different in others that they are considered to be 2 different and separate identities.

Since the writers found no record in literature of a *Physalospora* pathogenic in any Ericaceous plant it is thought that the blueberry canker fungus has not been previously described and named. Since the fungus insofar as is known at present inhabits principally the cortical tissues of canes and shoots, a name to indicate that characteristic is being applied with the following description:

Physalospora corticis n. sp.

Perithecia mostly scattered, partly imbedded, black, globose to conical, ostiolate, about 200–350 μ high and 170 to 210 μ wide; asci 8-spored, clavate, wall thick especially at the apex, hyaline, 112–180 μ long and 27–32 μ wide; paraphyses erect, anastomosing, filamentous; ascospores hyaline to slightly tinted, ellipsoid to fusoid, continuous, 24–37 μ long and 9.6 to 16 μ wide.

Pycnidia scattered, black, sub-globose to conical, wall thick, 90–208 μ high and 60–165 μ wide. Pycnospores hyaline, non-septate, fusiform elliptical, 27–45 μ long and 8–12 μ wide and produced on conidiospores about 15 μ long.

Small hyaline non-septate globose to rod-shape micropycnospores about 3–7 μ long, sometimes present with pycnospores or in separate pycnidia.

Parasitic in stems and shoots of blueberries (*Vaccinium australe* Small and *V. ashei* Reed), causing cankers, in Alabama, Florida, Georgia, North Carolina, and Mississippi.

Perithecia plerumque conspersa, partim immersa, atra, globosa vel conica, ostiolata, 200–350 μ alta, 170–210 μ lata; asci octospori, clavati, tunica praesertim apice incrassata, hyalini, 112–180 μ longi, 27–32 μ lata; paraphyses rectae, anastomosantes, filamentosae;

ascosporae hyalinae vel pallide coloratae, ellipsoideae vel fusoideae, continuae, 24–37 μ longae, 9.6–16 μ latae.

Pycnidia conspersa, atra, subglobosa vel conica, pariete crasso, 90–208 μ alta, 60–165 μ lata; pycnosporae hyalinae, continuae, fusiform-ellipticae, 27–45 μ longae, 8–12 μ latae; e conidiophoris circa 15 μ longis productae; micropycnosporae parvae, hyalinae, continuae, globosae vel bacilliformes, circa 3–7 μ longae, pycnosporis associatae vel pycnidiis separatis interdum praesentes.

Hab. cancros in caulibus surculisque Vacciniorum producens, in Alabama, Florida, Georgia, North Carolina, et Mississippi.

A type specimen of the fungus has been deposited in the Mycological Collections of the Bureau of Plant Industry, U. S. Department of Agriculture, at Washington, D. C., under No. 71,375. This specimen was collected in a North Carolina cultivated field of *Vaccinium australe*.

CONTROL

Spraying blueberry bushes with Bordeaux mixture and with lime-sulphur solution in both winter and summer has given inadequate control of the disease.

Growers have attempted to hold the disease under control by pruning out all visible cankers, even taking out badly infected bushes, but this practice has proved of little value. A great majority of the conspicuous cankers can be removed, especially if they are on the secondary shoots, but it is difficult to train workmen to recognize the more incipient stages where evidence of the disease is in the form of small reddish conical swellings or unevenness of the bark.

The most promising lines of attack are the planting of disease-free plants and the use of resistant varieties. Selection of disease-free plants for transplanting will not always guarantee freedom from the disease in some southern sections, as the wild blueberries in nearby swamps and forests are sometimes infected and the disease may spread from them to cultivated fields. Therefore, at present, the best recommendations for combating blueberry canker is to grow only those varieties known to be resistant. Cabot and Pioneer are very susceptible varieties, and, if planted in the South, they should not be near infected fields or in localities where there is wild-blueberry growth.

SUMMARY

This paper discusses a fungus disease of considerable economic importance prevalent in southeastern United States, causing extensive and damaging cankers on blueberry stems and shoots.

The disease has been observed in cultivated plantings in Alabama, Florida, Georgia, Mississippi, and North Carolina, and in wild blueberry plants in Florida and North Carolina. Evidence indicates that the causative fungus is indigenous on wild blueberries in the South and has spread from them to cultivated plantings.

The fungus is thought to be an undescribed species of *Physalospora* and the name **P. corticis** n. sp. is proposed.

U. S. DEPARTMENT OF AGRICULTURE,
BUREAU OF PLANT INDUSTRY STATION,
BELTSVILLE, MARYLAND.

PHYTOMONAS POINSETTIAE N. SP., THE CAUSE OF A BACTERIAL DISEASE OF POINSETTIA

MORTIMER P. STARR[1] and P. P. PIRONE[2]

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The poinsettia, *Euphorbia pulcherrima*, probably is the most important potted plant for the Christmas market. Heretofore, the most serious disease of this plant has been a stem rot of rooted cuttings and of young plants caused by a species of *Rhizoctonia*. A new and much more destructive disease, caused by a bacterium, has been under investigation since July, 1941. The first recorded outbreaks of this disease occurred in two widely separated greenhouse establishments in New Jersey during the summer of 1941; by November, it was known to occur in Maryland, Pennsylvania and New York. Preliminary studies of this disease were made by the junior author and his assistant (7) who also first isolated the bacterium herein conclusively proved to be the specific etiological agent.

DESCRIPTION OF THE DISEASE

Symptoms are readily visible in all above-ground plant parts. Longitudinal, water-soaked streaks, usually on one side of green stems, are the most characteristic symptoms. Such streaks may continue up through the leaf petioles, resulting in spotting or blotching of the leaves and in complete defoliation. The streaking may continue downward into the woody stem, detectable only by a bark incision. A yellowing of the cortex and browning of the vascular system is revealed when the epidermis is removed above the water-soaked lesions on green stems. In advanced stages, the stems crack open in a very unsightly manner, and bend down sharply towards the unaffected side.

Glistening, golden-brown masses of bacteria occasionally ooze from the ruptured stems and from leaf lesions. Microscopic sections of invaded stems show bacteria in profusion in the phloem.

Cuttings obtained from infected stock plants fail to develop into desirable plants. In one greenhouse these, as well as the stock plants, were a total loss within a month after the disease was first observed.

ISOLATION AND PROOF OF PATHOGENICITY OF THE CAUSAL ORGANISM

The causal organism was first isolated by the junior writer from diseased stock plants on July 25, 1941. The surface of young green stems showing typical longitudinal streaks was wiped with 95 per cent alcohol and the epidermis peeled off. Dilution plates prepared from the yellowed cortical tissues thus exposed yielded a single kind of bacterial colony. Early in August, isolates from these plates were inoculated by means of needle pricks

[1] Bacteriological Laboratory, Brooklyn College, Brooklyn, New York. Contribution 47 from the Department of Biology.

[2] Agricultural Experiment Station, New Brunswick, New Jersey.

into 12 young shoots on 4 vigorous plants just above the leaf axils. Within 3 weeks unmistakable lesions appeared on the stems. In some instances these had progressed sufficiently to involve the leaves and stem tips above the inoculated areas. Within 5 weeks after inoculation, the stems cracked open and the exposed tissues became brown and dry in much the same way as the naturally infected stems. Control plants receiving only needle pricks and sterile nutrient broth remained healthy throughout the course of the work. Bacteria, reisolated from these artificially infected plants, were morphologically similar to those inoculated.

Two of the original isolates (CP 1 and CP 2)[3] were sent to W. H. Burkholder, Cornell University, who, on September 5, 1941, succeeded in causing typical symptoms by needle-pricking the young stems of potted poinsettias with these two isolates. Uninoculated, but wounded, controls remained healthy. Bacteria (CP 3 and CP 5),[3] morphologically similar to those inoculated, were isolated November 25, 1941, by the senior writer from lesions of these artificially infected plants.

On October 15, 1941, the senior writer isolated similar bacteria (CP 13 and CP 14)[3] from lesions of a naturally infected diseased poinsettia that had been forwarded by W. H. Burkholder. Ten isolates from this plant were inoculated into wounds made with the tip of a sharp, sterilized scalpel on the young stems and petioles of 10 healthy poinsettias. Characteristic symptoms appeared about the sites of inoculation on all 10 plants within 4 weeks; within 6 weeks, the stems had cracked open. Bacteria (CP 20 and CP 21)[3] resembling those that had been applied as inoculum were isolated from the advancing margins of the lesions from 3 to 20 weeks after inoculation. Wounded, noninoculated control plants remained healthy throughout these experiments.

Another series of isolations (CP 27, 31, 35, 37, 39 and 41)[3] was made by the junior writer on November 10, 1941, from diseased stock plants originally obtained from the greenhouse, where the disease was first noted in July, but had been held over in the interim in both the plant pathology and ornamental horticulture greenhouses at New Brunswick. Isolations were attempted from 16 stems on 6 stock plants nearly 4 months after these plants first showed typical symptoms. These were made from brown longitudinal streaks beneath unruptured stems, from brown subsurface lesions, just beyond the ruptured stem areas, and from deep discolored lesions originating at stem ruptures. In nearly all cases, a species of bacterium, morphologically resembling that obtained in the original isolations was recovered. Three isolates were inoculated November 17, 1941, on 6 stems in a manner similar to that already described. Well-defined lesions did not appear on these stems until 5 weeks after inoculation. The delay in appearance of symptoms may have been due to the lower greenhouse temperatures prevailing at that time. Adequate controls remained healthy. Bacteria similar to those inoculated were isolated from typical lesions.

[3] The isolates designated in parentheses are the 14 upon which is based the following description of the pathogen.

CONTROL SUGGESTIONS

It is suggested that stock plants known to harbor the pathogen be discarded, even though the new shoots used for propagation appear normal. Rooted cuttings and young plants obtained from questionable sources should be kept separate from those known to come from healthy stock. Overhead watering and syringing should be minimized to avoid spreading the bacteria, which ooze from infected stems and leaves. It may be of interest to note that healthy plants were grown successfully in the greenhouse involved in the July 25th outbreak, following complete removal and burning of all diseased plants, steam sterilization of the soil and disinfection of the benches, pots and other greenhouse apparatus. It is not definitely known whether these measures were entirely responsible for the apparent control.

DESCRIPTION OF THE PATHOGEN

A representative group of isolates[4] from naturally and artificially infected poinsettias was studied in detail by the senior writer. The technique used in this study is that described in the *Manual of Methods for Pure Culture Study of Bacteria* (9) and in the *Manual of Dehydrated Culture Media and Reagents* (4). Where the procedure used is not exactly as described in these publications, appropriate particulars of the technique are given or other sources are cited.

Except where indicated otherwise, incubation of cultures was carried out at 27° C. The names of colors used in the descriptions follow those suggested by Ridgway (8). In all cases, 14 isolates[4] were studied simultaneously and, unless stated otherwise, identical reactions were obtained for all the isolates.

Morphology and Staining Reactions

Highly pleomorphic: straight rods, comma-shape, curved, coccoid, clavate, wedge-shape, and bizarre involution forms, occurring singly and in palisade arrangement. Barred and granule-bearing forms common. Endospores not formed. Capsules formed in some sugar-containing media. Motile by one, rarely two, polar or lateral flagellum. Size very variable: 0.2–0.8 × 0.5–8.5 μ, averaging 0.3–0.6 × 1.0–3.0 μ. Gram-positive, becoming Gram-variable, as shown by Hucker's modification of Gram's stain. The Gram-negative cells in old cultures often contain Gram-positive granules; these granules are also disclosed by staining with Loeffler's alkaline methylene blue. Not acid-fast by the Ziehl-Nielsen method. Stained readily by carbol-fuchsin and malachite-green. Cells are not stained by Congo red or nigrosin. Flagella of cells grown on moist beef extract agar slants of pH 6.9 are stained easily by Gray's method.

Cultural Characteristics

Colonies on Beef-extract Agar (0.3 per cent beef extract, 0.5 per cent peptone, 1.5 per cent agar; pH 6.9). Surface colonies are round, slightly convex, ranging from 0.1 to 1.0 mm. and averaging 0.2 to 0.5 mm. in diameter; deep colonies are ellipsoidal. Amorphous or finely granular structure detectable at a magnification of 440×; edges are entire at 100×. Smooth, non-viscid, moist, colorless, almost transparent.

Colonies on Potato-dextrose Agar (Bacto; pH 5.6). Surface colonies are round, slightly convex, ranging from 0.5 to 4.0 mm. in diameter; deep colonies are ellipsoidal, 0.1–0.3 × 0.3–0.8 mm. Finely granular structure evident at 440×; edges entire at 100×. Smooth, glistening, non-viscid, mucoid, moist, salmon color becoming ochraceous salmon.

Colonies on Tryptose-phosphate Agar (Bacto tryptose phosphate broth plus 1.5 per

[4] See footnote 3.

cent agar; pH 7.3). Surface colonies are round, convex, 4 mm. or more in diameter; deep colonies are ellipsoidal averaging 0.4 × 1.5 mm. Finely granular structure visible at 440×; edges entire at 100×. Glistening, smooth, mucoid, moist, frequently becoming very viscid, carrot-red or flesh color; deep colonies are colorless.

Colonies on Blood Agar (Bacto-blood agar base plus 5 per cent sterile, defibrinated rabbit blood; pH 6.9). Surface colonies round, convex, under 1 mm. in diameter. Structure amorphous at 440×; edges entire at 100×. Glistening, moist, slightly viscid, white or pale-salmon. No visible action on blood.

Colonies on Tellurite-blood Agar (Bacto-dextrose-proteose No. 3 agar plus 5, 8 or 10 per cent bacto-tellurite-blood solution; pH 7.4). Surface colonies round, convex, 0.3 to 2.5 mm. in diameter. Amorphous structure, entire edges, smooth, glistening. Light to deep neutral-gray, rarely pale or pallid neutral-gray. Tellurite reduced.

Beef-extract Agar Slants (pH 6.9). Scanty, filiform, glistening, non-viscid, colorless, almost transparent.

Potato-dextrose Agar Slants. (Bacto; pH 5.6). Moderate, filiform, glistening, non-viscid, salmon or flesh color.

Tryptose-phosphate Agar Slants (Bacto-tryptose-phosphate broth plus 1.5 per cent agar; pH 7.3). Abundant, filiform, later spreading, raised, glistening, mucoid, often becoming extremely viscid, orange-pink to geranium-pink.

Potato Slants. Abundant, spreading, not raised, glistening, moist, becoming butyrous or somewhat viscid, orange, Mikado orange, deep chrome- or capucine-yellow. Potato distinctly browned in ten days.

Loeffler's Blood-serum Slants (Bacto; pH 7.2). Moderate, filiform or spreading, glistening, non-viscid, carnelian-red. Medium liquefied in 3 to 10 days.

Agar-shake Cultures (2 per cent tryptose, 0.5 per cent yeast extract, 1.5 per cent agar). Growth only under distinctly aerobic conditions in upper 1 to 2 mm.

Thioglycollate Broth (BBL thioglycollate medium (1) with dextrose and Eh indicator; pH 7.4). Flocculent pellicle and dense, viscid growth in upper 2 or 3 mm. and no trace of growth in anaerobic medium below, thus indicating a distinct partiality for aerobic conditions.

Litmus Milk (pH 7.0). Only very slight acidity or no visible change for 1 to 2 weeks after inoculation; then, soft coagulation, reduction of litmus, followed by a rapid, complete peptonization.

Lead Acetate Agar (Bacto; pH 6.6). Moderate auburn surface growth, extending 1 to 2 cm. along line of stab as a scanty beaded growth. Medium not browned, except slightly after 3 or 4 weeks; it is questionable whether this slow, scanty browning should be interpreted as a definite production of hydrogen sulphide, especially since hydrogen sulphide cannot be demonstrated by means of lead acetate paper suspended in tryptone-broth cultures, even after 10 weeks' incubation.

Beef-extract Broth (0.3 per cent beef extract; 0.5 per cent peptone; pH 6.9). Turbid within 24 hours, later viscid, pale-salmon sediment; no pellicle or ring formed.

Tryptose-phosphate Broth (Bacto; pH 7.3). Turbid within 24 hours, later, abundant flesh-pink, viscid sediment; sometimes, ring and pellicle.

Tryptone Water (1 per cent Bacto tryptone; pH 7.0). Turbid within 24 hours, later, viscid sediment; no pellicle or ring.

Range of Temperatures for Growth. Active cultures were transferred to small tubes of Bacto-tryptose phosphate broth of pH 7.3 previously brought to the given temperature, incubated there, and examined daily for the definite turbidity that indicates growth. After 24 hours, growth occurs between 15 and 36° C. and, after 48 hours, between 7 and 12° C. No growth after a week from 0.5 to 5° C. or from 37 to 50° C.

Carbon Metabolism

Utilization of Sodium Citrate as Sole Carbon Source. Koser's citrate medium (Bacto; pH 6.7) does not support growth.

Utilization of Glucose as Sole Carbon Source. A synthetic medium consisting of 0.1 per cent $NH_4H_2PO_4$, 0.02 per cent KCl, 0.02 per cent $MgSO_4 \cdot 7H_2O$, 1.0 per cent glucose, pH 6.4, does not support growth, even after 8 weeks.

Action on Cottonseed Oil. No lipolytic activity revealed by spirit-blue cottonseed-oil-agar technique (10, 11).

Voges-Proskauer and Methyl-red Tests (Bacto M.R.–V.P. medium; pH 6.9). No acetylmethylcarbinol can be demonstrated in 72-hour-old cultures by the Werkman (12) procedure. Methyl-red test is also negative.

Sugar Fermentations. The ability to ferment carbohydrates, alcohols, and glycosides was determined by means of Durham tubes with a medium consisting of 0.3 per cent yeast extract, 0.5 per cent peptone, 0.5 or 1.0 per cent of the sugar, pH 7.5, with either 1 per cent Andrade's indicator or 0.0018 per cent phenol red to demonstrate acid production. As is indicated below, acid is produced from many sugars; gas is not formed.

No acid produced after three weeks: Rhamnose,[5] fucose,[6] inulin,[5] glycogen,[6] mannitol,[5] dulcitol,[5] sorbitol[6] inositol[6] (except isolates CP 27, 31, and 41, which ferment inositol slowly).

Weak, slow acid production: Arabinose[5] (9 days), xylose[5] (9 days), lactose[6] (2 to 3 weeks; acid production from lactose cannot be demonstrated, even after 6 weeks, when Andrade's indicator is used), trehalose[6] (12 days), dextrin[5] (8 days), adonitol[6] (9 days).

Moderate to abundant acid production within 5 days: Glucose[6] (when Andrade's indicator is used, there is demonstrable only a slow and variable production of acid from glucose), fructose,[5] mannose,[5] galactose,[5] sucrose,[5] maltose,[5] cellobiose,[6] melibiose,[6] raffinose,[6] glycerol,[5] erythritol,[6] salicin,[5] amygdalin.[6]

Hydrolysis of Starch (0.3 per cent yeast extract, 0.5 per cent tryptone, 0.5 per cent "soluble" starch). Complete hydrolysis after 11 days, but not after 4 days, as shown when portions of the cultures are tested with Lugol's iodine solution on a spot plate.

Action on Cellulose. No visible action on strips of filter paper immersed in beef-extract broth cultures for 8 weeks.

Nitrogen Metabolism

Indole Production. No indole formed in 1 per cent Bacto tryptone of pH 7.0 after 3, 6, 9 or 15 days, as determined by the Ehrlich-Böhme procedure.

Reduction of Nitrates. No reduction of 0.1 per cent $NaNO_3$ in beef-extract broth of pH 7.0 in six days, as shown by the sulphanilic acid-α-naphthylamine test.

Urea Hydrolysis. No hydrolysis of a 1 per cent aqueous-urea solution by heavy suspensions of 3-day-old cells during an incubation of 5 hours at 37° C., as determined by a negative Nessler test for ammonia. This procedure can demonstrate ureolysis by *Bacillus pasteurii* and ureolytic micrococci after an incubation period of only 1 hour at 37° C.[7]

Utilization of Uric Acid as Sole Nitrogen Source. Koser's uric acid-glycerol medium (6) does not support growth.

Utilization of Asparagin as Sole Source of Both Carbon and Nitrogen. A synthetic medium consisting of 0.1 per cent KH_2PO_4, 0.02 per cent KCl, 0.02 per cent $MgSO_4 \cdot 7H_2O$, 0.5 per cent asparagin, pH 6.4, does not support growth.

Action on Gelatin (0.3 per cent beef extract, 0.5 per cent peptone, 12 per cent Bacto gelatin; pH 6.6). Definite crateriform liquefaction in 3 days at 23° C.; later, stratiform liquefaction extending down 2 to 4 cm in 2 weeks at 23° C.

Hydrolysis of Sodium Hippurate (0.3 per cent yeast extract, 0.5 per cent tryptone, 0.5 per cent sodium hippurate). After 7 days' growth, benzoic acid was sought by adding either 1 volume of 7 per cent $FeCl_3$ or of 50 per cent H_2SO_4 to 4 volumes of culture and looking for a precipitate of ferric benzoate or of benzoic acid, respectively. No hydrolysis of sodium hippurate is demonstrated by this technique, although a known hippurate-splitting species, *Streptococcus agalactiae,* does bring about demonstrable hydrolysis.

TAXONOMY OF THE PATHOGEN

The monotrichic phytopathogenic bacteria are now included in the genus *Phytomonas* Bergey *et al.* The poinsettia pathogen, described above, is unlike any now included in that genus in the fifth edition of *Bergey's Manual of Determinative Bacteriology* (2); hence, the name **Phytomonas poinsettiae** n. sp., is proposed.

Burkholder (2, 3) and others have discussed the heterogeneous nature of the genus *Phytomonas* Bergey *et al.,* and have shown that, actually, it consists of several unrelated groups of bacteria. It seems likely, on the basis of characteristic morphological and cultural properties, that this poinsettia pathogen is closely related to those Gram-positive, phytopathogenic bacteria, now (2) in Appendix II of the genus *Phytomonas* Bergey *et al.,* some of which have been more correctly included (5, 8a, 11) in the genus *Corynebacterium* Lehmann and Neumann. When the latter genus is emended to include motile forms, this species might be included as *Corynebacterium poinsettiae.*

[5] Andrade's fuchsin indicator was used for demonstrating acid production.
[6] Phenol red was used for indicating production of acid.
[7] Starr, Mortimer P. Unpublished observations, 1940.

SUMMARY

A serious disease of the poinsettia, *Euphorbia pulcherrima,* is described. A bacterium, proved beyond all question to be the specific etiological agent of this disease, is described in detail. Since the causal agent is different from all previously reported species of phytopathogenic bacteria, the name **Phytomonas poinsettiae** n. sp., is proposed for this pathogen, and it is recommended that this species be included in the *Corynebacterium* group of plant pathogenic bacteria.

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BORON DEFICIENCY IN PEAR TREES

J. R. KIENHOLZ

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Boron has been applied to pear orchards in an attempt to correct various troubles of the fruit and tree. A cracking and dieback of pear in New South Wales (1, 2) has been corrected by soil applications of borax. Noble (10) reports a blossom and twig blight in the same country, which appeared to be associated with boron deficiency. A roughening and cracking of Bosc pears responded to boron treatments in Tasmania (3), but it was pointed out that "crinkle" was not affected by the treatment. McLarty (9) illustrated boron deficiency symptoms on unnamed pear varieties in British Columbia, but Eastham (6) reported that drought spot of Bosc pears was unaffected by boron applications in the western part of the Province. One report from Washington (13) suggested that the incidence of a hard end and cork-spot complex of Anjou and Bartlett pears may have been reduced by boron treatments.

BORON TREATMENTS ON PEAR TREES IN OREGON

A type of pitting on Bosc pear fruits was noticed in 1937 at Hood River, Oregon. The pitting differed from that caused by the stony-pit virus (8), although the two were often associated on the same tree. Following 1937, the Bosc trouble increased in severity; fruit cracking appeared, less leaf surface developed, production decreased, and considerable dieback was evident by 1940. A similar trouble on Bartlett pears also was seen in 1937, but, following boron applications to this orchard by the grower, the trouble did not reappear. The same malady was again found on this variety in a small irrigated orchard at The Dalles, Oregon, in 1938, and at Hood River in 1941.

In 1940 a small uniform block of Bartlett pear trees was selected at The Dalles for boron treatment. These trees were approximately 12 years of age, and had produced only pitted fruits in 1939. Twelve ounces of granular borax was applied to the soil about the trees on February 9, 1940. Fruit failed to set in 1940 because of a lack of proper pollination and late frosts. The effect of the treatment was apparent in the 1941 crop, however, and results as recorded on August 6 of that year are given in table 1.

It will be observed (Table 1) that boron corrected the pitted condition of the fruit. The higher average yield per tree of the treated trees appeared to be caused by elimination of a blighting of blossoms prevalent in the plot of untreated trees.

Individual trees of the Bosc variety at the Hood River Experiment Station were selected for treatment in 1941. The trees were of different ages and located in various parts of the pear orchard. It had been observed in previous years that they produced pitted or cracked fruits and that dieback of the twigs had developed on several of them. One pound of granular

TABLE 1.—*Effect of boron applications on Bartlett pear pitting*

Type of treatment	Trees in plot	Sound fruits 1941	Pitted fruits 1941	Average fruits per tree, 1941
	Number	*Number*	*Number*	*Number*
Checks—no boron	10	201	472	67.3
12 oz. borax per tree, Feb. 9, 1940	14	1255	0	89.6

borax was applied to the soil about each tree on March 7, 1941. The amount of pitted fruit produced the season before and the season immediately after treatment is shown in table 2.

TABLE 2.—*Effect of boron applications on Bosc pear pitting and cracking*

Type of treatment	Trees in plot	1940		1941		Average fruits per tree, 1941
		Sound fruits	Pitted fruits	Sound fruits	Pitted fruits	
	Number	*Number*	*Number*	*Number*	*Number*	*Number*
Check trees—no boron	6	1186	115	1652	327[a]	330
1 lb. borax per tree, Mar. 7, 1941	8	220	942	3424	0	428

[a] Records taken Sept. 1, 1941.

It is evident that boron applications eliminated the fruit pitting. Half of the treated Bosc trees bore fruit with stony-pit symptoms both before and after treatment, thus substantiating former experience that stony pit is not responsive to boron treatments (8).

SYMPTOMS OF BORON DEFICIENCY ON BOSC AND BARTLETT PEARS

Observations during the past 5 years indicate that pear trees are less sensitive to boron deficiency than most apple varieties. From the limited material available for observation it also appears that more variation in the symptoms may be expected among pear varieties. Of the 3 leading commercial varieties grown in Oregon, the susceptibility to boron deficiency in descending order appears to be: 1. Bosc, 2. Bartlett, and 3. Anjou. The specific symptoms of the deficiency on the first 2 varieties may be summarized as follows:

On the Fruit. Circular to angular, blunt bottomed, shallow depressions (4–10 mm. in diameter by 2 mm. or less in depth) may be present on the fruit surface. They are more abundant toward the calyx end and often merge into an extended area resembling certain forms of the hard- or black-end disease. The color of the pitted surface and of the tissue within and immediately surrounding each pit, often remains darker green than adjacent tissue. A small central core of corky tissue underlies each depression and tissue adjacent to this cork rapidly becomes brown upon exposure to air.

The calyx region may be pointed, sunken, or somewhat lopsided, but usually has the general appearance of the disease known as hard end. (This feature is less evident in the Bosc variety.) The calyx tissue is invariably hard when cut. Masses of brown, corky cells are generally present under the calyx lobes and may extend well into the core area. Mild to severe cracking of the fruit may accompany the shallow pitting of Bosc pears. The form of the pits and the corky calyx region serve to distinguish this trouble from most other types of pitting found on pear fruits (Fig. 1).

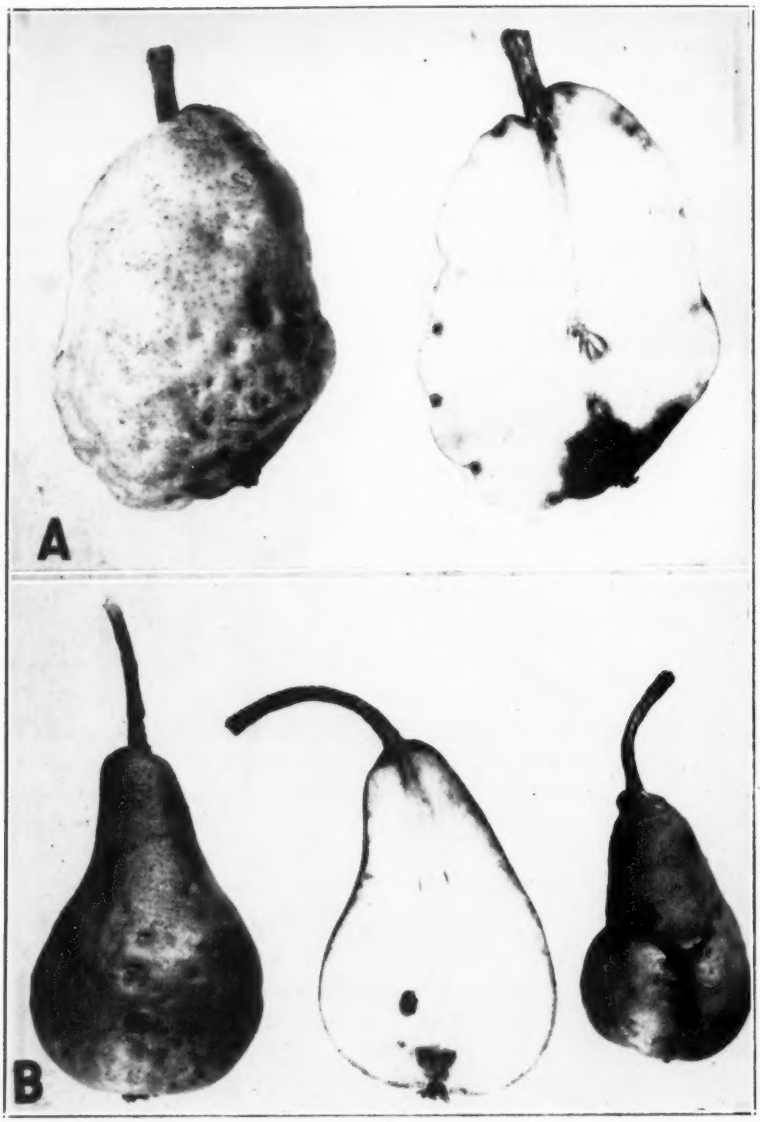

FIG. 1. Boron deficiency symptoms on Bartlett pear fruits (*A*) and Bosc pear fruits (*B*).

On the Tree. Irregular, superficial, dark or light colored bark cankers with slightly raised, uneven borders may appear on younger branches. These borders tend to give a somewhat flattened appearance to the affected parts. Fewer basal leaves develop on diseased twigs; eventually 1 or 2 sets of terminal, dwarfed leaves may form the total leaf surface. A dieback of the twigs follows and the tree gradually dies, unless the deficiency is corrected. Symptoms on the tree may develop without fruit symptoms, or only poorly defined symptoms may accompany the fruit pitting.

STONY PIT IN RELATION TO BORON

More experience is needed to distinguish between the true boron-deficiency and stony-pit symptoms on different pear varieties. Cork or crinkle

of pears failed to respond to boron treatments in Tasmania (5), but a roughened and cracked condition of the fruits was corrected (3). Since stony pit is known to occur in British Columbia on Bosc pears, Eastham (6) may have been dealing with the virus trouble. Stony pit often is called drought spot by fruit growers. An illustration of the internal cork, bitter pit, and crinkle of Bosc pears as they occur in Australasia (4, Fig. 4) leaves little doubt that they are in reality stony pit. The varieties listed as susceptible to cork by Carne and Martin (5) are of interest also, since several of them have been found to be susceptible to stony pit in Oregon. Bartlett has been proved to be a symptomless carrier of the stony pit virus at Hood River.

Insufficient material of Anjou pears showing boron-deficiency symptoms has been examined by the writer to distinguish boron deficiency with certainty from stony pit. From the limited material observed, however, it appears that fruit symptoms for both troubles may be quite similar, and this may be true for other varieties, as well. Sufficient evidence has accumulated to warrant the statement that boron has no direct influence in correcting the stony pit disease. Since stony pit symptoms may become partly masked, or vary in intensity during different seasons on the same tree, sufficient trees should be observed to evaluate the variation in these symptoms.

BLACK END AND BORON TREATMENTS

The disease of pears commonly known as hard end or black end is usually attributed to the grafting of commercial varieties on oriental rootstocks (7). A report from Washington (13) suggested that boron treatments may have reduced the incidence of this and a cork spot of Anjou and Bartlett pears. The symptoms of cork or drought spot in Anjou pears were previously described from Washington (11, 12). They bear some similarity to those of stony pit as it has been observed on Anjou pears in Washington and Oregon.

There is some evidence that several hard-end or pit conditions of unknown origin may appear in pear fruits. Some of these may occasionally respond to boron treatments. The types of hard- or black-end attributed to stock and cion relations, however, have not been corrected with boron applications. A well kept Anjou orchard that had produced considerable worthless fruit affected with black end in past years was selected for treatment at Hood River. The trees were over 25 years of age, remarkably uniform in appearance, and on *Pyrus serotina* rootstocks. Besides the 3 test plots, an additional 5-acre tract treated by the grower was kept under observation. The experimental plots consisted of check trees left untreated, trees sprayed with borax (25 pounds in 300 gallons of water) at the delayed dormant period, and trees that received borax as a soil application in early spring. The results of these treatments are given in table 3.

The fruit was severely affected by black end in 1938, only slightly affected in 1939, and moderately so in 1940. Development of black end was not correlated with boron treatments, and varied considerably from year to year. Analyses of selected terminal leaves indicate that boron passed from the

TABLE 3.—*Effect of boron treatments on black end of Anjou pears*

Type of treatment 1939	Trees in plot	Black end infected fruit			Boron in leaves, August 1939[a]
		1938	1939	1940	
	Number	*Per cent*	*Per cent*	*Per cent*	*p.p.m.*
Check trees—no boron	27	100	1.7	90	20.4
1 lb. borax per tree on soil, Mar. 7	11	100	1.8	95.9	31.3
Borax spray—(25–300) on trees, Mar. 24	24	100	1.5	96.2	27.7

[a] Analyses by L. P. Batjer. Dr. Batjer stated that greater differences probably could be expected from analyses of fruit samples.

roots to the terminals through the cion unions on these trees growing on roots of a species known to be associated with the black end condition.

SUMMARY

Boron applications to pear trees corrected a condition characterized by pitting of fruits, especially near the calyx end, and by superficial cankers on younger branches. A dieback of twigs follows, and the tree gradually dies, unless the deficiency is corrected.

The symptoms of boron deficiency on fruit are similar to those of stony pit, a virus disease.

It is suggested that the disease of pear variously termed crinkle, bitter pit, and internal cork in Australasia is the same as stony pit.

The black end of pear fruits, attributed to stock and cion relations, was not corrected by applications of boron.

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VARIATION IN REACTION OF ANTHONY OATS TO STEM RUST, PUCCINIA GRAMINIS AVENAE[1]

IAN W. TERVET AND HELEN HART

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In July, 1939, numerous large pustules of stem rust, *Puccinia graminis avenae* Eriks. and Henn., appeared on a considerable number of plants of the resistant oat variety Anthony growing in test plots at the Crookston, Minnesota, Agricultural Experiment Station and in adjacent commercial fields. The same situation developed at Crookston in 1940, although there was very little stem rust on Anthony at the Waseca, Morris, Grand Rapids, and St. Paul stations. Anthony is no longer of commercial importance in Minnesota; but, since this variety has long been moderately to highly resistant to stem rust, it seemed desirable to ascertain whether a new physiologic race of *P. graminis avenae* had appeared or whether the unusual development of rust might have been due to the effects of extraordinary weather or soil conditions or to varietal mixture in the oats.

All rust collections made from susceptible plants were identified by Wm. Q. Loegering[2] as *Puccinia graminis avenae*, race 5, to which Anthony always has been resistant as indicated by the production of small uredia surrounded by pronounced chlorotic or necrotic areas.[3] As the race 5 was consistently isolated from large uredia on susceptible plants, and as it is one of the races of oat stem rust most prevalent in the Mississippi Valley, it was clear that no new race was involved.

It seemed improbable that the susceptibility of Anthony at Crookston could have been due to ordinary varietal mixtures because the seedlots used for that station and the other stations previously mentioned had all been supplied by the central station at St. Paul. Samples of the rusted plants from Crookston were examined by H. K. Wilson, Division of Agronomy, University of Minnesota, who considered them Anthony plants on their gross morphological characters. Since the rust-susceptible plants were morphologically indistinguishable from the rust-resistant ones, it seemed possible that the unusual soil or climatic factors might have predisposed plants to rust.

To test the effect of meteorological conditions, seedlings of Anthony, grown from the 1939 seed lot[4] of University Farm and the same that was sent to Crookston for planting in 1940, were inoculated with race 5 of *Puc-*

[1] Assistance in the preparation of these materials was furnished by the personnel of the Work Projects Administration, Official Project No. 165–71–1–124, sponsored by the University of Minnesota. Paper No. 1990, the Scientific Journal Series, Minnesota Agricultural Experiment Station.

[2] Agent, Bureau of Entomology and Plant Quarantine, United States Department of Agriculture.

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[4] Seed lot supplied by H. K. Wilson, Division of Agronomy, University of Minnesota.

cinia graminis avenae under varying conditions of light, temperature, atmospheric moisture, and soil moisture; but there were no consistent differences in rust development under the various conditions. Occasional plants, however, became heavily rusted in all the tests. The tests were repeated several times, with the same results: there was no association between particular environmental conditions and the number of abundantly infected plants. The number of susceptible Anthony plants produced by seedlots grown at the several experiment stations of the University of Minnesota, however, varied somewhat. In one experiment, of 20 seedlings from seed grown at Waseca in 1938, 15 plants were susceptible and only 5 resistant. Seed lots from other branch stations, nevertheless, did not produce so many susceptible plants; and of a total of 550 seedlings inoculated, only 16 per cent were susceptible.

Because of the lack of positive information from the greenhouse tests and because susceptible plants of Anthony were found in the field only at

Diagram 1. *The arrangement of fertilizer plots for Anthony oats at Crookston and at St. Paul, Minnesota, in 1941*

Phosphate Boron	Phosphate	Phosphate Manganese	Phosphate Zinc	Phosphate	Phosphate Boron Manganese Zinc
Boron	No treatment	Manganese	Zinc	No treatment	Boron Manganese Zinc
Phosphate Potash Boron	Phosphate Potash	Phosphate Potash Manganese	Phosphate Potash Zinc	Phosphate Potash	Phosphate Potash Boron Manganese Zinc

Crookston, experiments on the possible effect of fertilizers were made at Crookston and St. Paul in 1941. The trial plots included fertilizer treatments with treble superphosphate at 100 lb. an acre, treble superphosphate at 100 lb. an acre plus potash at the same rate, and 3 minor-element treatments with boron at 15 lb., manganese at 25 lb., and zinc at 25 lb. an acre. Plots were arranged as in diagram 1, treatments being made separately and also as combinations of fertilizers and minor elements.

So little stem rust, only 5 per cent, developed in Crookston plots in 1941 that no differences were observable. At St. Paul 15 per cent stem rust developed, but there were no differences either in plant development or in stem-rust infection in the different plots. However, about 15 per cent of the plants were severely rusted (50 to 60 per cent) at St. Paul and between 10 and 15 per cent in the plots at Crookston; and, again, race 5 of *Puccinia graminis avenae* was identified from 4 collections from the St. Paul plots and 2 collections from the Crookston plots. The susceptible plants at St. Paul were examined by H. K. Wilson, Division of Agronomy, University of Minnesota, who considered them indistinguishable from Anthony.

Susceptible and resistant seedlings of Anthony that had been inoculated with race 5 in the greenhouse were transplanted to the field and grown to maturity. The seedling reaction (Fig. 1) was maintained in the adult plants, the susceptible seedlings giving rise to plants with numerous large pustules, the resistant seedlings to plants with small uredia and indications of necrosis. There was 50 per cent rust on the susceptible plants.

Seed was saved from susceptible and resistant plants in the above tests and, similarly, from susceptible and resistant plants in the fertilizer tests. Seedlings grown from this seed were inoculated in the greenhouse with race 5 of *Puccinia graminis avenae* collected from susceptible Anthony at Univer-

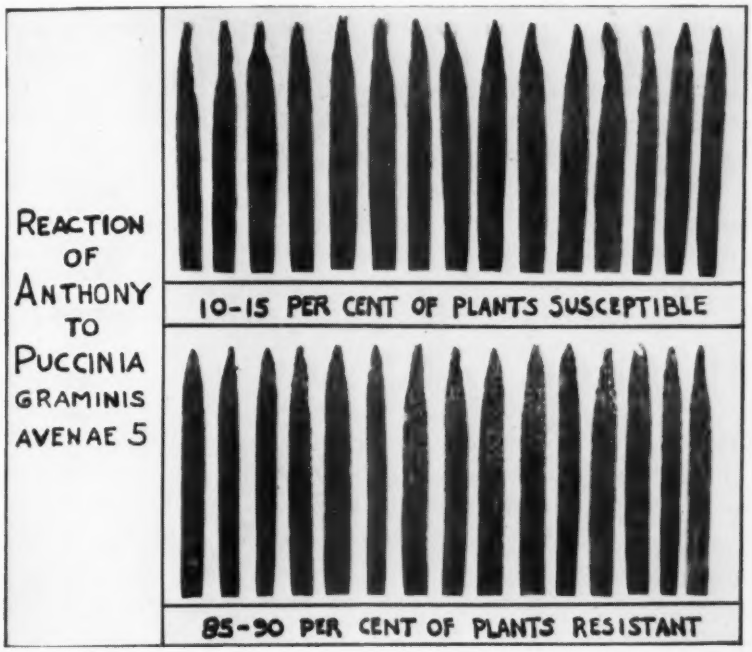
REACTION OF ANTHONY TO PUCCINIA GRAMINIS AVENAE 5
10-15 PER CENT OF PLANTS SUSCEPTIBLE
85-90 PER CENT OF PLANTS RESISTANT

FIG. 1. The reactions of seedlings of Anthony oats (C. I. 2143) to race 5 of *Puccinia graminis avenae* and the proportions of susceptible and resistant plants in field plots in Minnesota.

sity Farm, St. Paul, in 1941. In all cases seed from susceptible plants produced susceptible seedlings and that from resistant plants, resistant seedlings.

It appears, therefore, that there are at least 2 strains of Anthony oats, indistinguishable morphologically but differing in reaction to stem rust race 5, (Fig. 1). The percentage of susceptible plants in the seedlots tested is about 10 or 15 per cent. About 15 per cent of a total of 500 adult plants were susceptible in field plots at St. Paul. There is no doubt that the seed lots of Anthony oats in use in Minnesota contain many plants susceptible to at least one of the prevalent races of oat stem rust.

Spectrographic analyses[5] were run on the resistant and susceptible

[5] We are indebted to Richard C. Nelson, Philip Hamm, and David Gottlieb for the spectrographic analyses.

Anthony seedlings to see if they differed in ability to absorb from the soil certain elements that might affect reaction to rust. Densitometer readings for the magnesium and iron lines for the 2 strains of Anthony were nearly identical, and inspection of the lines for other emission elements on the film revealed no differences between the resistant and susceptible plants.

In conclusion it seems advisable in the case of varieties that have been bred for resistance to disease to practice periodic reselection within the variety. Resistant varieties should be exposed to artificial epidemics of diseases to make possible the observation of any variation in disease reaction and to eliminate segregates or, even, mutants that are not pure for the resistant character. Strains are known to exist within other cereal varieties and to differ with respect to disease reaction. Renown wheat was not pure for resistance to leaf rust, *Puccinia rubigo-vera tritici* (Eriks. and Henn.) Carl., but reselection within this variety produced a highly resistant strain.[6] The hard red winter wheat, Crimean C. I. 1435, is considered by Kiesselbach and Peltier[7] a mass variety; they demonstrated that the majority of 578 of its most promising strains fell into 3 different groups on the basis of their reaction to selected races of *P. graminis tritici* Eriks. and Henn. In contrast to our experience with Anthony oats, many of the strains of Crimean differed from each other agronomically, as well as in rust reaction. However, in Kanred, a stem-rust resistant selection from Crimean, Kiesselbach and Peltier found a few individuals susceptible to a race of stem rust that, ordinarily, did not attack Kanred. Johnston[8] also noted the existence within supposedly pure wheat varieties of strains indistinguishable morphologically but differing in reaction to disease. He worked with approximately 200 soft red winter wheat varieties that were ordinarily susceptible to race 9 of leaf rust, *P. rubigo-vera tritici;* and in 28 of them he found strains resistant to leaf rust. Thus he believed that selection within a variety was an excellent means of obtaining rust resistant strains. And from another standpoint, it is probable that many disease-resistant crop plants need periodic reexamination, exposure to artificial epidemics of disease, and reselection within the variety to ensure the maintenance of its resistance.

UNIVERSITY FARM, ST. PAUL, MINN.

[6] Newman, L. H., J. G. C. Fraser, and A. G. O. Whiteside. Handbook of Canadian spring wheat varieties. Canadian Dept. Agr. Farmers Bull. 18. (Publ. 538). 1939.

[7] Kiesselbach, T. A., and G. L. Peltier. The differential reaction of strains within a variety of wheat to physiologic forms of *Puccinia graminis tritici.* Neb. Agr. Exp. Stat. Res. Bull. 39. 1926.

[8] Johnston, C. O. The occurrence of strains resistant to leaf rust in certain varieties of wheat. Jour. Amer. Soc. Agron. 21: 568–573. 1929.

PHYTOPATHOLOGICAL NOTES

New Fungicides and Reduced Fungicide Dosages for the Control of Kernel Smut of Sorghum.—During the 1942 season, four new fungicidal seed treatment materials and several previously used were tested for their effect on emergence in sorghum, and on the control of covered kernel smut at full, half, and fourth dosages.

The following materials were used: New Improved Ceresan, 5 per cent ethyl mercuric phosphate (Bayer Semesan Co.); copper carbonate, 54 per cent metallic copper; Spergon, 99 per cent tetra-chloro parabenzoquinone (U. S. Rubber Company); Thiosan (DuBay 1205–FF), 50 per cent tetramethyl thiuramdisulphide; and DuBay 870, 100 per cent ferric dimethyl dithio carbamate (Bayer-Semesan Company); Captax, 100 per cent mercaptobenzo-thiazole (R. T. Vanderbilt Company); Sanoseed, 2.2 per cent ethanol mercuric chloride (Ansbacher Siegle Corporation); M.T.D.S., morpholine thiuram disulphide (M. C. Goldsworthy); and dusting sulphur (Stauffer Chemical Company).

Seed of Sharon kafir was dusted with spores of covered kernel smut at the rate of 1 g. of spores to 100 g. of seed; separate portions were then treated with New Improved Ceresan at the rates of $\frac{1}{2}$, $\frac{1}{4}$, and $\frac{1}{8}$ oz. per bu., and with the other dusts at 3, $1\frac{1}{2}$, and $\frac{3}{4}$ oz. per bu.

The treated seed, along with similarly smutted untreated seed, was planted at Beltsville, Maryland, at the rate of 200 seeds per 44-foot row, replicated 4 times for each rate of application of each fungicide. Similar plantings were made at 7 other stations in the Midwest and Southwest, 3 replications per location.

Data on emergence were obtained at 5 stations and on smut control at 7. At Hays and Manhattan, heavy rains caused irregular emergence; at Tucson, high temperature inhibited smut development. The results are summarized in table 1.

On the whole, Thiosan, DuBay, 870, copper carbonate, and Spergon seemed most consistently beneficial to emergence, while Sanoseed and sulphur were of little, if any, benefit in this respect. Spergon controlled smut perfectly Thiosan and DuBay 870 reduced it to an average of less than 0.1 per cent at all dosages, while M.T.D.S. and copper carbonate were only slightly less effective. Captax and sulphur were fairly effective at full dosage, and were superior to New Improved Ceresan, which was 2 years old and evidently had deteriorated. Sanoseed was highly ineffective in smut control.

Smut in the untreated checks ranged from 7.8 to 43.4 per cent and averaged 25.2 per cent and hence the fungicides were not put to a uniformly severe test. However, the excellent smut control obtained with the new materials, Thiosan, DuBay 870, and M.T.D.S. at all dosages, and with Captax at the maximum dosage, indicates their possible effectiveness in controlling certain diseases of other field crops. If so, they can be substituted for those

TABLE 1.—*Effect of seed treatment with different fungicides at three rates of application, on seedling emergence of and on occurrence of covered kernel smut on Sharon kafir at several stations, 1942*

Treatment		Percentage emergence[a] and percentage heads smutted at												Totals and averages			
Fungicide	Oz. per bu.	Beltsville, Md.		Lincoln, Nebr.		Garden City, Kans.		Stilwater, Okla.		Dalhart, Tex.		Manhattan, Kans.	Hays, Kans.	Av. Emer.	Heads		
		Emer.	Smut	Emer.	Smut	Emer.	Smut	Emer.	Smut	Emer.	Smut	Smut	Smut		Total No.	Smutted	
																No.	Pct.
New Improved Ceresan[b]	½	54	1.8	70	3.3	73	1.6	69	0.3	61	2.4	1.0	0.0	65	2088	34	1.63
	¼	57	11.2	69	8.1	70	4.9	82	1.8	57	3.5	2.5	1.6	67	2266	120	5.30
	⅛	57	9.8	50	20.6	65	11.2	92	3.2	55	12.4	11.2	1.1	64	2164	195	9.01
Copper carbonate	3	59	0.0	78	0.0	71	0.7	69	0.0	67	0.0	0.0	0.0	69	2300	3	0.14
	1½	61	0.0	74	0.0	73	0.5	86	0.0	59	1.3	2.0	0.0	71	2173	8	0.37
	¾	58	0.7	58	0.7	74	0.5	80	0.0	63	0.9	0.0	0.0	67	2126	10	0.47
Spergon	3	62	0.0	54	0.0	78	0.0	85	0.0	60	0.0	0.0	0.0	68	2410	0	0.0
	1½	60	0.0	63	0.0	73	0.0	85	0.0	69	0.0	0.0	0.0	70	2416	0	0.0
	¾	57	0.0	49	0.0	70	0.0	94	0.0	56	0.0	0.0	0.0	65	2410	0	0.0
Thiosan	3	64	0.0	75	0.0	69	0.0	80	0.0	68	0.0	0.0	0.0	71	2486	0	0.0
	1½	63	0.0	74	0.0	68	0.0	93	0.0	60	0.0	0.0	0.0	72	2162	0	0.0
	¼	53	0.2	64	0.0	63	0.0	88	0.0	59	0.0	0.0	0.0	65	2221	1	0.05
DuBay 870	3	68	0.4	77	0.0	65	0.0	83	0.0	66	0.0	0.0	0.0	72	2505	2	0.08
	1½	59	0.4	72	0.0	70	0.0	90	0.0	65	0.0	0.0	0.0	71	2407	2	0.08
	¼	55	0.2	53	0.0	61	0.0	91	0.0	65	0.3	0.0	0.0	65	2189	2	0.09
Captax	3	58	0.0	66	0.0	71	0.5	81	0.0	61	0.0	0.0	0.0	67	2218	2	0.09
	1½	55	2.9	59	1.2	63	0.8	79	0.0	62	0.6	0.7	0.0	64	1985	21	1.06
	¾	51	9.1	53	5.1	58	1.9	70	1.4	58	3.5	5.3	0.5	58	1815	73	4.02
Sanoseed	3	57	13.2	58	13.4	52	6.1	80	4.8	54	13.2	11.9	6.9	60	2119	207	9.77
	1½	46	25.6	54	26.3	52	18.9	81	9.2	50	17.8	38.4	0.0	57	2010	365	18.16
	¾	45	17.3	43	43.2	54	12.7	86	9.8	47	28.6	35.3	5.6	55	1956	360	18.40
M.T.D.S.	3	57	0.9	59	0.0	57	0.3	77	0.0	63	0.0	0.0	0.8	63	2101	7	0.33
	1½	59	0.2	62	0.0	62	0.0	85	0.0	57	0.0	0.0	0.0	65	2077	1	0.05
	¾	54	2.2	63	0.0	52	0.8	86	0.0	53	0.3	0.0	1.0	62	2011	15	0.75
Sulphur	3	44	2.3	71	0.0	51	0.3	77	0.0	44	0.3	0.0	0.5	57	1995	10	0.50
	1½	51	1.3	59	3.9	50	1.5	85	0.0	45	0.0	0.0	0.0	58	1882	19	1.01
	¾	50	1.2	61	0.0	47	2.6	82	0.0	52	0.3	1.9	0.0	58	2156	28	1.30
None	.....	49	34.4	64	33.5	57	12.9	78	13.0	51	25.2	31.1	7.8	60	1969	442	22.45
	.....	48	40.7	56	31.5	57	25.8	73	15.0	40	29.5	15.9	10.5	55	1718	471	27.42
	.....	52	33.3	61	43.4	51	20.3	77	17.3	45	25.9	25.4	11.7	57	2013	519	25.78

[a] Emergence data were not obtained at two stations because of washing.
[b] Made in 1940 and apparently deteriorated.

fungicides containing metals highly essential in the manufacture of armaments. Thiosan is on the market as a fungicide for turf, while Captax is used in the manufacture of rubber. DuBay 870 and M.T.D.S. are still in the experimental stage. R. W. LEUKEL,[1] Bureau of Plant Industry Station, Beltsville, Md.

Epichloe typhina on Imported Fescue Seed.—In a search for stoloniferous fescues suitable for airfields, fairways, and playing surfaces, the department of agronomy at this station imported seed of two strains of *Festuca rubra genuina* and one of *F. rubra nemoralis* from Hungary in 1939, as follows:

Strain 1. *F. rubra genuina* via T. W. Woods & Sons, Richmond, Virginia
" 2. *F. rubra nemoralis*" " " " " " " "
" 3. *F. rubra genuina* " Benö Bálint & Sons, Budapest, Hungary

Seed was planted in flats in the greenhouse and approximately 250 individual plants of each strain were transferred to the nursery for observation and subsequent selection. Strain 3 showed the greatest variation in growth habits and types but individual plants from all strains were chosen for further study. These plants were divided to give 20 plant clones and transferred to another location for further observation on growth habit, seed yield, foliage color, disease resistance, etc. In June of the current year these clones were in their second season of seed production.

Of 104 selections of strain 3, 27 clones showed this year fruiting structures of *Epichloë typhina.* In a few clones every seed stalk had the typical sign of the "choke" disease, bearing white stromata on the leaf sheaths and occasionally on some of the spikelets of the panicle. Often the panicle failed to emerge. In other clones at least one plant showed evidence of infection.

According to Sampson and Westen[1] this fungus is systemic in *Festuca rubra* and is transmitted through the seed in percentages as high as 99; infected plants clonally divided eventually show disease in every plant of the clone. Plants systemically infected may produce a hundred or more panicles, yet show nothing of the parasite to the casual observer.

In lieu of this information all infected clones were removed from the nursery and burned. A few badly infected plants were removed to an isolated plot and interplanted with noninfected plants in an effort to establish rate of spread under field conditions. Seed from these infected plants was collected for the purpose of studying seed transmission and the effect of hot-water treatment on seed disinfection. It is doubtful if these studies will yield pertinent information until three years hence.—C. C. WERNHAM, Dept. of Botany, The Pennsylvania State College, State College, Pa.

[1] The generous cooperation of the following investigators at these stations is gratefully acknowledged: J. E. Livingston and R. L. Cushing at Lincoln, Nebr., A. E. Lowe at Garden City, Kans., E. G. Heyne, at Manhattan, Kans., A. F. Swanson at Hays, Kans., J. B. Sieglinger and D. E. Hoffmaster at Stillwater, Okla., A. T. Bartel at Tucson, Ariz., and B. F. Barnes at Dalhart, Tex.

[1] Sampson, K., and Western, J. H. Diseases of British grasses and herbage legumes, 26–29. 1941. Cambridge Univ. Press.

[a] Emergence data were not obtained at two stations because of washing.
[b] Made in 1940 and apparently deteriorated.

An Apple Rot Fungus Morphologically Related to a Human Pathogen.—A rot of stored apples, attributed to *Sporotrichum malorum* by Kidd and Beaumont,[1] was brought to the attention of the writer in 1937, when this disease was observed causing serious losses in stored Winesap apples in Virginia. Kidd and Beaumont,[1] in England, reported it as new in 1924, and named the causal fungus *Sporotrichum malorum*. Gardner[2] reported that what he considered a strain of the same fungus caused a decay of Grimes Golden apples in Indiana in 1929. In 1931 Ruehle[3] in a report of "New Apple Rot Fungi from Washington" listed *Sporotrichum malorum* Kidd and Beaumont and also described a new species, which he designated as *S. carpogenum*.

Since 1937 the writer has isolated similar fungi from decaying apples, from cankerous apple wood, and from the surface soil beneath apple trees. Instead of finding the isolates to be either identical with or distinctly different from the published species, the writer observed a wide range of variation in the 130 single-spore isolates studied. Preliminary comparison of the isolates with a sub-culture of *S. malorum* obtained from the Centraal Bureau vor Schimmelcultures indicated that the original classification was in error. The present study, therefore, has been made in an effort to determine the proper systematic position of the fungus described by Kidd and Beaumont as *Sporotrichum malorum*, and that described from a similar apple rot by Ruehle as *S. carpogenum*, and to find the relationship between these fungi and the many variable isolates obtained during the course of this study.

It has been demonstrated that this fungus has dark pigment in the hyphae and to a certain degree in the spores; therefore, it should be placed in the Dematiaceae instead of in the Moniliaceae. Since the process of sporulation is characterized by a continuous proliferation of the sporogenous cells, forming conidia through a collar-like apex, where they abscise but are held together in a mucous mass, it seems evident that this fungus does not belong in the form-genus *Sporotrichum*. It, therefore, is being transferred to the form-genus *Phialophora*, and *Sporotrichum malorum* Kidd and Beaumont becomes *Phialophora malorum* (Kidd and Beaumont) comb. n. *Sporotrichum carpogenum* becomes synonymous with *P. malorum* and is considered to be a strain of that species.

The form-genus *Phialophora*, as established by Thaxter (in Medlar),[4] is characterized by spore formation in cups borne on the tips of flask-shape conidiophores. *P. verrucosa* was designated as the type species and at that time was the only species of the new form-genus. This fungus had been isolated from a skin disease, reported as the first case of *dermatitis verrucosa*

[1] Kidd, M. N., and A. Beaumont. Apple rot fungi in storage. Trans. Brit. Mycol. Soc. **10**: 98–118. 1924.

[2] Gardner, Max W. Sporotrichum fruit spot and surface rot of apple. Phytopath. **19**: 443–452. 1929.

[3] Ruehle, George D. New apple rot fungi from Washington. Phytopath. **21**: 1141–1152. 1931.

[4] Medlar, E. M. A new fungus, *Phialophora verrucosa*, pathogenic for man. Mycologia **7**: 200–203. 1915.

(*chromoblastomycosis*), a skin disease of man. The apple-rotting fungus aligns itself with the form-genus *Phialophora,* strictly in a morphological sense, because of the unique conidial formation. There has been no evidence of any relationship between the diseases that these fungi cause. Apples inoculated with *P. verrucosa* remained sound, and, even under semi-saprophytic conditions, the fungus did not grow or cause any disintegration of the apple tissue. In 1937 Conant[5] concluded that the form-genus *Cadophora,* with its specific forms reported as causing bluing and staining of wood pulp, is synonymous with the form-genus *Phialophora.* Inasmuch as *Phialophora* had priority, he proposed new combinations for the described species. The transfer of the apple-decaying species with its numerous strains to *Phialophora malorum* is a step further in showing the wide distribution of this form-genus in nature.—L. P. MCCOLLOCH, Bureau of Plant Industry Station, Beltsville, Md.

Willow Blight in West Virginia.[1]—In 1941 Leach and Rupert[2] reported willow blight in West Virginia near Thomas in Tucker County. It was observed only on trees of an ornamental planting. The owner stated that the trees had been obtained from an out-of-State nursery, but the exact origin could not be determined, as the records of purchase had been lost. The disease has since been found in limited areas on native willows in two adjacent counties, but it is not widely distributed. All infections on native willows apparently are of relatively recent origin, as there are few dead twigs or old cankers. It seems probable that the disease has spread to native willows from the planted stock.

The frequent association of *Physalospora miyabeana* Fuk. and *Fusicladium saliciperdum* Tub. in willow blight has led to some dispute as to which is the primary parasite. The disease was first reported in this country by Clinton[3] and Clinton and McCormick,[4] who regarded *F. saliciperdum* as the causal agent. In England Nattrass[5] and Dennis[6] regarded *P. miyabeana* as the primary parasite and relegated the *Fusicladium* to the rôle of a secondary invader. Harrison[7] in Canada and Brooks and Walker[8] in England concluded that both organisms are responsible for injury.

[5] Conant, Norman F. The occurrence of a human pathogenic fungus as a saprophyte in nature. Mycologia **29**: 597–598. 1937.

[1] Published with the approval of the director of the West Virginia Agricultural Experiment Station as Scientific Paper No. 293.

[2] Leach, J. G., and J. A. Rupert. Black canker of willow in West Virginia. U. S. Dept. Agr. Pl. Dis. Rptr. **25**: 588. 1941.

[3] Clinton, G. P. A new disease of willows appears in Connecticut. U. S. Dept. Agr. Pl. Dis. Rptr. **11**: 87–88. 1927.

[4] Clinton, G. P., and F. A. McCormick. The willow scab fungus. Conn. Agr. Exp. Stat. Bull. 302. 1929.

[5] Nattrass, R. M. The Physalospora disease of the basket willow. Trans. Brit. Mycol. Soc. **13**: 286–304. 1928.

[6] Dennis, R. W. G. The black canker of willows. Trans. Brit. Mycol. Soc. **16**: 76–84. 1931.

[7] Harrison, K. A. Willow blight. Canada Dept. Agr. Rept. Dom. Bot. (1928): 34–36. 1929.

[8] Brooks, F. T., and M. M. Walker. Observations on *Fusicladium saliciperdum.* New Phytol. **34**: 64–67. 1935.

When the disease was first observed in West Virginia the season was well advanced and only *Physalospora miyabeana* was observed associated with blighted twigs, but in May, 1942, *Fusicladium saliciperdum* was found fruiting abundantly on infected petioles. No acervuli of the *Physalospora* were present then, although the fungus could be isolated from the cankers. In June, acervuli of *P. miyabeana* were found to be very active, while the *Fusicladium* was definitely on the decrease. Perithecia of *P. miyabeana* were found in some of the cankers in late summer and in the winter.

Pure-culture inoculations showed the *Physalospora* to be pathogenic on both stems and leaves of growing trees, in the greenhouse and out of doors, as well as on excised leaves in a Petri dish. The *Fusicladium*, however, under comparable conditions, failed to infect the stems and leaves of growing trees. On excised leaves it produced only a weak, delayed infection after the leaves had begun to deteriorate.

In nature there was always a sudden appearance of a saprophytic species of *Cladosporium* on the affected leaves very soon after they began to die. It is probable that the *Fusicladium* may act somewhat similarly, although there are some indications that it may be weakly parasitic. However, in these experiments it was not nearly so virulent as *Physalospora*.

A species of *Macrophoma* was occasionally found fruiting in old cankers and dead twigs but this fungus was never isolated from blighted leaves or twigs in the early stages of infection. It, also, is most likely a secondary invader.—JOSEPH A. RUPERT and J. G. LEACH, Department of Plant Pathology and Bacteriology, West Virginia University, Morgantown, W. Va.

Longevity of the Spores of Some Wood-destroying Hymenomycetes.—Fresh sporophores of each fungus were placed in a moist chamber for 12 to 24 hours to deposit their spores on clean glass slides. These spore prints were allowed to dry at room temperature before wrapping in tissue paper for storage at room temperature. At approximately weekly intervals spores were sown on slants of Sabouraud's dextrose agar. A No. 30 platinum wire with a loop about 1 mm. in diameter was flame-sterilized. A drop of sterile water picked up in this loop was used to rinse the spores from a small area on the slide. The spores were streaked out on the agar slant, 3 such loopfuls being transferred to each tube. The moistened area on the spore print was marked on the reverse of the slide with wax pencil, and thereafter avoided in securing spores for further sowings. After the spot on the slide was dry, the latter was returned to its tissue-paper wrapping.

The tube was capped with a double thickness of wax paper and held at room temperature. Growth was observed with the help of a 10 × hand lens. With proper care to obtain spores from healthy sporophores, bacterial or mold contaminations were rare.

Stereum hirsutum spores were alive after 56 but not after 64 days of storage. Growth was distinctly visible at the end of the first week.

Stereum rugisporum spores were alive after 46 but not after 66 days.

At least two weeks were required for visible growth. The sowings made after 16 and 38 days failed to show growth.

Stereum sanguinolentum spores were alive after 131 days but not after 137 days. Growth was visible after 4 or 5 days. Only one earlier sowing, that after 101 days, failed to grow.

Polyporus schweinitzii spores were alive after 162 but not after 170 days. Growth was visible after 2 or 3 weeks.

Fomes igniarius spores were alive after 91 but not after 99 days. Growth was visible after 20 to 27 days. Germination was somewhat erratic, fresh spores and those stored 7, 14, and 64 days failing to germinate.

Fomes pinicola spores were alive for at least 173 days. Growth was visible after 5 to 7 days.

Trametes pini spores were alive for at least 65 days. About 2 weeks were required for growth to become visible.

Polyporus abietinus spores were alive for at least 65 days. About 1 week was required for growth to appear.

Pleurotus ostreatus spores were alive at least 20 days. Growth was visible in about 1 week.

In an earlier experiment, *Coniophora sistotremoides* spores grew when sown on casein-glucose-potato agar after 46 but not after 68 days. At first growth appeared in 17 or 18 days, but the last two successful sowings required about 50 days to make visible growth.

Hymenochaete tabacina spores grew on walnut nutrient agar after being dry for 17 days. Growth was visible in 4 or 5 days.

These rather fragmentary results are reported at this time, since the work is necessarily suspended for the duration of the war.—CHARLES H. HARRISON, 2318 N. 38 Street, Seattle, Washington.

BOOK REVIEWS

KARLING, JOHN S. (Columbia University). *Plasmodiophorales.* 144 p., 17 pl., 17 figs. Publ. by the author (New York) 1942.

Had this book emanated from a government bureau it would doubtless have been seized upon by a Congressional committee investigating useless expenditures as an example of the sort of activity that must be suppressed in time of war, notwithstanding that it contains some references to diseases of economic crops. On the other hand, it is likely to be hailed by mycologists who have not completed their transfer of allegiance to Mycology's domineering offspring, Phytopathology, as another volume in the series of American monographs that illuminates minutely a corner of the realm of fungi for the sake of knowledge itself—a series distinguished by such names as Thaxter, Burt, Fitzpatrick, Coker, and Couch. The present work exhibits less of the author's personal handiwork than some of its predecessors but is still an authoritative treatment of a field in which specialists are rare.

Its scope is roughly indicated by the table of contents as (1) Introduction, (2) Cytology, (3) Sexuality and alternation of generations, (4) Classification and description of species, (5) Phylogeny and relationships, and (6) Diseases caused by species of the group. In the discussion of "promitosis" and the "akaryote stage" the author stresses the finding by recent authors, employing improved technique, of chromosomes in the vegetative divisions, and their failure to find convincing evidence of an akaryote condition. Thus these features, which have been held to relate the Plasmodiophorales to the Myxomycetes and Protozoa, are gravely suspected of not occurring in the group at all.

The author has consistently maintained a judicial attitude toward the evidence, often fragmentary, on the cytology and life cycle of the organisms that different investigators have assigned to this group, and the evidence in figures and in text is carefully reviewed, even when the claim to relationship is rejected. He recognizes 8 valid genera, as compared with 5 by Fitzpatrick in *The Lower Fungi*, 1930, but 2 of these have been described since the latter book appeared. He regards the group as belonging to the primitive fungi with its closest affinity to the Woroninaceae, though of questionable relationship to the other families usually included in the Chytridiales. In postulating this relationship the presence of biflagellate heterocont zoospores in the Plasmodiophorales is emphasized. He does not view the evidence regarding sexuality and alternation of generations in the group as warranting general acceptance. The reviewer feels that the chapter entitled Introduction is more helpfully read at the end since it provided a needed synthesis after he had lost his bearings in navigating the many details of the 4 ensuing chapters. He also found it helpful, in gaining a quick conspectus of the group, to add symbols to the index of species on p. 137 differentiating the valid genera and species from the others; a slight modification of typography would have gained the same end.

The final chapter gives very full information on club root of crucifers and powdery scab of potatoes, including (for the former) a complete host list, a table of effects of various fungicides and crop ameliorants on the pathogen, and lists of varietal reactions. Separate bibliographies pertaining to the subject matter of each chapter are provided. The illustrations are numerous and well executed.—FREEMAN WEISS.

CHESTER, K. STARR. *The Nature and Prevention of Plant Diseases.* I–XII, 1–584. The Blakiston Company. 1942. $4.50.

The publication of a new textbook in plant pathology immediately raises the question as to the reason for its publication. Is it the result of a definite need for something better; and if so, does the new text supply the need? In the preface of this new book, the author states that one of his objectives is to provide the student with a work to which he may refer for detailed and specific directions on plant-disease control; and follows with a statement that "I have tried to select for detailed study diseases that are of considerable economic importance over a broad area of the United States . . . and that illustrate the leading principles of plant pathology." Also an attempt is made to "rectify the past neglect of diseases of southern and prairie crops."

The author seems to have met these objectives fairly successfully. The selection of subjects is good and should meet general approval, particularly the discussion of a larger number of diseases caused by the imperfect fungi. The arrangement of the text is somewhat different from that of some other texts, but not radically so. It begins with a chapter on "The significance of plant disease in agriculture." This is rather a thrilling statement of the losses farmers sustain year after year as a result of outbreaks of diseases in their crops. At the same time, the author works in a very good limited history of plant pathology. In the second chapter, types of plant diseases are outlined, with the remainder of the chapter devoted to a discussion of the fungi, their classification and types of fruiting bodies, and infection by fungi. The next 7 chapters are devoted to diseases caused

by the fungi, the arrangement being as follows: Basidiomycetes, Rusts; Smuts; Fleshy fungi and Mycorrhizae; Ascomycetes; Imperfect fungi; Phycomycetes; and Damping-off and related troubles. These are followed by chapters on Bacterial diseases; Virus diseases; Parasitic seed plants and algae; Nematodes; Physiogenic diseases; Methods of studying plant diseases; Environment and parasitic disease; Etiology and epiphytology of disease; and three chapters on principles and procedures in the control of plant diseases: 1, by regulation, 2, by inducing resistance, and 3, by cultural methods. This is followed by a rather complete index.

The author has discussed the majority of the most destructive or, at least, the better-known plant diseases and has also included in each group a number of minor diseases; for example, 21 rusts are treated, including rusts of grasses, forage crops, trees and shrubs, vegetables, and ornamentals. The diseases are given space according to the economic losses they cause. Seventy-two pages are devoted to the diseases caused by fungi imperfecti, with a discussion of 24 well-selected diseases. A feature of the chapter on damping-off and related troubles is the discussions of disease complexes, as the cotton-seedling blight and boll-rot complex; corn root, stalk, and ear rots; and sorghum root and stalk rots. The author is at his best in the field with which he is most familiar, namely, the diseases caused by viruses. If the text is widely adopted, a great impetus will have been given to the use of binomials in naming viruses, because it is accepted without reservation. In the chapter on physiogenic disease, less than a page is devoted to the general symptoms produced in plants by a shortage of N, P, and K. Without headings it would be difficult for one experienced in nutrient deficiencies to guess which deficiencies were being described. Photographs of typical nutrient deficiencies would add greatly to the value of this part of the discussion.

A successful textbook in plant pathology will be the chief source of information on plant diseases for numerous students, both in class and in the field later on, and for an occasional one it will form the ground work upon which he will build his knowledge of plant diseases as a profession. Each group has a right to expect, in a text, a high degree of scholarship with as nearly absolute accuracy as the literature will provide; and where all of the facts are not known, a high degree of judgment in the interpretation of the available facts. While the text is a well-arranged, well-balanced discussion of plant diseases for the beginner, yet, an occasional mistake makes one lose a certain degree of confidence not only in the discussion in which it occurs but in the discussion of those topics with which one is not too familiar. For example, the author has the "double-sexed mycelium" of stem rust, following fertilization of the receptive hypha in the pycnium, "growing vigorously down into the barberry leaf," and feeding "on its cells" (p. 38); and teliospores "fall to the ground," resist winter temperatures, and the following spring germinate, etc. (p. 39). It is doubtful whether the scab fungus invades the corn stem and causes barrenness; or that in the southern States the corn root-rot stage (caused by the scab fungus) is common (p. 108); or that the powdery mildew fungi produce toxins that pass through the leaf tissue and yellow and kill cells (p. 135). The powdery mildew epidemic of red clover in 1922 is commented upon as though the fungus were of long standing in this country, but flared up suddenly that year and then subsided, rather than pointing out that it was a new introduction at about that time from Europe and has been injurious each year since its introduction (pp. 136, 452). What is meant by the statement that the sexual overwintering stage of *Sclerotinia fructicola* was determined in 1909? Surely not first discovered and described that year (p. 138). In speaking of the peach leaf-curl fungus the statement is made that "it oversummers and overwinters as ascospores on the bud scales," and "in the leaf curl fungi saprogenesis is wanting in nature but may be experimentally produced on culture media." Actually, there seems to be no evidence that the ascospores play any necessary part whatever in the life history of the fungus, once it is established in a peach orchard; but the yeast-like saprophytic stage appears to perpetuate the fungus year after year (pp. 147, 466). In control of the Texas root-rot fungus, it is claimed that, on small areas, soil disinfectants, such as 1.25 per cent formaldehyde or 0.25 per cent organic mercury, may be used to eradicate the fungus, but the disinfectant must penetrate to a depth of 4 feet. Actually, formaldehyde is completely filtered out of solution in passing through about 4 inches of soil (p. 179). The spread of the peach scab-fungus during the growing season usually is attributed to water rather than "by means of wind-blown conidia" (p. 196).

The false but widespread belief that southern anthracnose has been the principal limiting factor in red clover production in Tennessee and Kentucky and surrounding States is repeated by the author, although it is not supported by the literature. According to the usually accepted definition, *Phyllosticta solitaria* would be considered a facultative saprophyte and not a facultative parasite (p. 212). The same mistake is made elsewhere in the text. Sweet-potato ring rot is not caused by *Rhizopus nigricans* but by *Pythium ultimum* (p. 237). It is questionable whether bruised fruits and vegetables, held in an environment favorable to the fungus, *invariably* develop *Rhizopus* infection. The toxin secreted by the wildfire organism does not seem to be responsible for the necrotic reaction

in tobacco, as is claimed (p. 284). There seems to be a fair consensus of opinion that zinc-lime spray does not control bacterial spot of peach, but does reduce arsenic injury (p. 296). While it may be true that "alfalfa wilt is the most important disease of the crop," the statement would be definitely misleading to students in certain alfalfa-growing regions of the U. S. in which the disease rarely occurs (p. 310). The watermelon usually is considered nearly immune from the cucurbit bacterial-wilt pathogen and not susceptible, as the author's statement would imply (p. 312). It is rather the exception than the rule that "chlorophyll in leaves formed subsequent to inoculation" with a mosaic virus "is lacking in certain leaf areas." Chlorophyll may be suppressed to a certain extent but patterns are usually shades of green. It is not true that the tobacco mosaic "easily stands the heating involved in the curing process." In flue-curing, temperatures are commonly reached that destroy the virus completely in certain parts of the curing shed (p. 329). In the discussion of acquired immunity, ring-spot of tobacco is given as the classical example of a plant that recovers and develops immunity and shows no further symptoms. Pollen sterility and symptoms produced in "recovered" plants when the temperature is reduced are disregarded. "Immunity is absolute, a plant is immune or not immune from a pathogen," and immunity is defined as "freedom from disease because the qualities for development of the pathogen are lacking in the plant," and yet plants with acquired immunity all support the parasitic virus (pp. 330, 490). A millimicron does not, as the author claims, = 1 μμ, which is a micromicron or 1×10^{-12} meter. A millimicron = 1 mμ or 1×10^{-9} meter (p. 325). The statement that in ordinary tobacco no symptoms are seen on the inoculated leaf (tobacco-mosaic virus) will hardly stand scrutiny. While it may be true that several species of aphids spread ordinary tobacco mosaic, the evidence for this occurring in the field is extremely meager (p. 344).

The statement that *manganese* sulphate is recommended for control of *magnesium* starvation (p. 393) is evidently a typographical error, of which there are very few in the text (but on p. 481 *in* for *is;* p. 403 *preferable* for *preferably;* p. 203 *Dayton* for *Drayton;* p. 502 *of* for *or;* and p. 456 *pratctically*). It is doubtful whether it is correct to say that phosphorus shortens the vegetative period of plants. This certainly occurs when phosphorus is added to phosphorus-deficient plants, but it merely brings about normal growth. It is more nearly correct to say that phosphorus starvation lengthens the vegetative period (p. 456). What will the student of cold injury say to the statement that "tissues are frozen, the cells explode, and the plant is killed" (p. 409)? The statement that potatoes regularly produce true seed in the north should be qualified to agree with the facts (p. 413).

While the text is well thought out, and for the most part a well-written, well-illustrated book, the erroneous statements greatly detract from its value and raise the question as to whether it meets the high standards of scholarship that American plant pathologists have set up for themselves.—W. D. VALLEAU, Kentucky Agricultural Experiment Station, Lexington, Kentucky.

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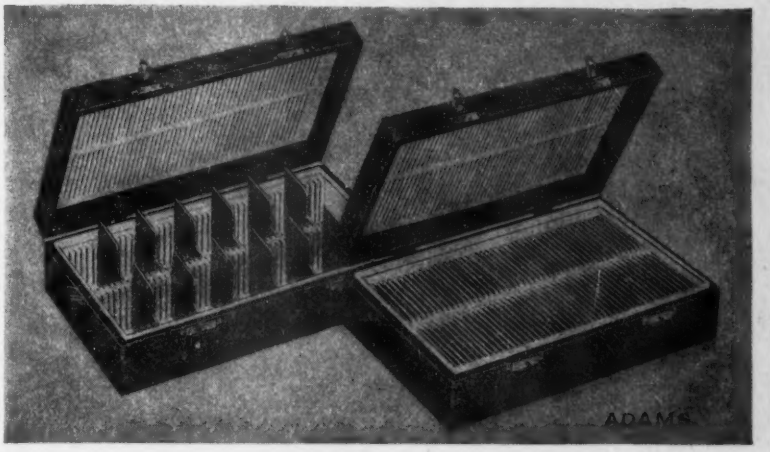

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